NEW ONSET SEIZURES IN ADOLESCENTS : A PROSPECTIVE OBSERVATIONAL STUDY

A THESIS SUBMITTED TO

BABA FARID UNIVERSITY OF
HEALTH SCIENCES, FARIDKOT (PUNJAB)

IN THE PARTIAL FULFILLMENT

FOR THE AWARD OF DEGREE OF

DOCTORATE OF MEDICINE

IN

PAEDIATRICS

MAY 2022

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I hereby declare that the thesis entitled "**New Onset Seizures in Adolescents : A Prospective Observational Study''** submitted towards partial fulfillment of the requirements for the award of MD in Pediatrics degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. JATINDER S GORAYA**, Professor, Department of Pediatrics and **Dr. Deepak Bhat**, Professor, Department of Pediatrics. No part of this thesis has formed the basis for the award of any degree previously.

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ACKNOWLEDGEMENT

I am highly grateful to the Almighty, without whose blessings no work can be conceived or completed. HIS presence we feel at each and every step of life. He's the one who hold our hands and guide us through everything.

I take this opportunity to thank everyone who have been helping me in the successful completion of this work.

With deep sense of gratitude I would like to convey my thanks to my esteemed teacher and supervisor Dr J.S.Goraya , Professor, Department of pediatrics, DMC&H Ludhiana, who has been most helpful and supportive throughout my tenure. His able and mature guidance, ever willing nature helped me to complete my task easily. He has always been an epitome of excellence for me.

It is with great reverence and profound pleasure that I acknowledge the valuable suggestions by my co-supervisor Dr Deepak Bhat, Professor, Department of Pediatrics, DMC&H Ludhiana .His constant encouragement during whole time helped me a long way in completion of this project.

I want to offer sincere thanks to Dr Puneet A Pooni, Professor and Head of Department of Pediatrics, DMC&H Ludhiana for her constant encouragement. I also wish to thank Dr Siddharth Bhargava, Dr Gurdeep Singh Dhooria, Dr Kamaldeep Arora, Dr Shruti Kakkar and Dr Karambir Singh Gill.

I am indebted to my senior Dr Sumati for her encouraging words, support and guidance throughout this duration of thesis .

I am also thankful to my seniors cum teachers Dr Akashdeep, Dr Alka, Dr Amita who taught me every intricacy of being a good pediatrician despite all odds.

My thanks to my colleagues Dr Harpreet, Dr Nikita, Dr Navotam, Dr Nikhar and Dr Disha for their brilliant company in this journey .They

have been invaluable support day in, day out for the entire work. Special thanks to my dear friends Dr Arpit and Dr Abhinav for their encouraging words and guidance throughout.

Words are woefully inadequate to express my sense of gratitude to my parents S. Gagandeep Bhutani and Smt Hema Bhutani for their love, selfless sacrifices, unfailing attention to my needs and blessings that have made me whatever I am today.

I am blessed to have Pranav Wadhwa as my husband to give his continuous support, encouragement and affection which is extremely valuable for me. I am also thankful to my gracious in laws for encouragement and their blessings.

Thanks to all the children who have been a part of this study and their parents as well. I sincerely thank the nursing staff of our department, their unending help made this uphill task possible.

Dr Tanvi

ABBREVIATIONS

AAN	American Academy of Neurology
ASS	Acute Symptomatic Seizures
CNS	Central Nervous System
CT	Computed tomography
EEG	Electroencephalogram
ILAE	International League against Epilepsy
GTCS	Generalised Tonic Clonic Seizures
MRI	Magnetic Resonance Imaging
NCC	Neurocysticercosis
US	Unprovoked Seizures

ABSTRACT

Background: New onset seizures can be acute symptomatic seizures caused by immediate brain insult or unprovoked seizures secondary to epileptic predisposition. New onset seizures have been well studied in children as well as adults but not in adolescents.

Aims and Objectives: To study clinical, electrographic, and neuroimaging characteristics of new onset seizures, and to determine their etiological profile.

Materials And Methods: Prospective observational study of adolescent children who presented with new onset seizures to the pediatric department of Dayanand Medical College & Hospital, Ludhiana from January 1, 2020 to June 30, 2021.

Results: Total of 51 adolescents, 40 boys and 11 girls, aged between 10-17 years were enrolled. Sixteen (31%) subjects belonged to acute symptomatic group and 35 (69%) to unprovoked seizure group. Presentation with generalized onset seizure was more common, observed in 11 (68.8%), and 22 (62.8%) patients, than with focal-onset seizures observed in 4(25%) and 10 (28.5%) patients, in the ASS and unprovoked seizure groups respectively. EEG changes were encountered only in the unprovoked seizure group and occurred in 14 (43%) of the 32 children. Neuroimaging abnormalities on CT and MRI were noted in 7 and 15 adolescents respectively. CNS infections accounted for 50%, head injury and metabolic derangements for 25% each of acute symptomatic seizures. Unprovoked seizures were largely caused by genetic or presumed genetic etiology in 23, structural etiology in 4 and unknown etiology in 8.

Conclusion: Acute symptomatic seizures account for one-third of the new onset seizures in adolescents and are mostly caused by neuroinfections, head injury and metabolic derangements. Unprovoked new-onset seizures are largely genetic in origin.

Keywords: New-onset seizure, acute symptomatic seizures, unprovoked seizures, epilepsy, adolescents.

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INTRODUCTION

A seizure is a sudden disruption of the brain's normal electrical activity resulting in transient occurrence of altered consciousness and/or other neurological and behavioural manifestations.[1] The term ‘seizure’ is derived from the Greek meaning ‘to seize’ or ‘to take hold’. Epilepsy, on the other hand is conceptually defined as, “a disorder characterized by an enduring pre-disposition to generate epileptic seizures and by neurobiological, cognitive, psychological and social consequences of this condition. The definition requires the occurrence of at least one epileptic seizure.[2] Epilepsy is not a unitary diagnosis but represents a diverse group of disorders, having in common an abnormally increased predisposition to seizures. The operational (practical) clinical definition epilepsy requires any of the following conditions-

i) At least two unprovoked (or reflex) seizures occurring at least 24 hours apart;
ii) One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over next 10 years;
iii) Diagnosis of an epilepsy syndrome. [2]

Classifications of seizure types and epilepsies have undergone a major change since its initial description in the 1960s.[3-6] The current (2017) International League Against Epilepsy (ILAE) classification[5] divides seizures into two major groups: focal onset or generalized onset, depending upon whether the initial manifestations of the seizure are focal or generalised (**Table 1**). Focal seizure is defined as “originating within networks limited to one hemisphere”[7]. Generalized seizure is defined as “originating at some point within and rapidly engaging, bilaterally distributed networks.[7] If the onset of seizure remains unknown, it is classified under unknown onset. Each of the major groups is further divided into motor and nonmotor types. In focal

seizures, level of awareness is used as a further classifier; focal onset with maintained awareness and focal onset with impaired awareness.[5]

Similarly, ILAE 2017 classification of the epilepsies[6] divides epilepsy into focal, generalised, combined generalised & focal, and unknown types (**Table 1**). Specific features such as age of onset, seizure type(s), EEG and neuroimaging findings may allow further classification of epilepsies into epilepsy syndromes[6]. From the causation perspective, epilepsies are classified as having structural, genetic, infectious, metabolic, immune and unknown etiologies. A further layer of comorbidities such as learning, psychological, and behavioural problems is added to the classification to emphasize not only their impact on persons with epilepsies but also to allow their early identification, diagnosis and treatment.[6]

Table 1: Classification of Seizure

<table>
<tr><td>FOCAL ONSET</td><td>GENERALISED ONSET</td><td>UNKNOWN ONSET</td></tr>
<tr><td>AWARE/IMPAIRED AWARENESS</td><td></td><td></td></tr>
<tr><td>MOTOR ONSET
AUTOMATISMS
ATONIC
CLONIC
HYPERKINETIC
MYOCLONIC
TONIC
EPILEPTIC SPASMS</td><td>MOTOR
TONIC-CLONIC
CLONIC
TONIC
MYOCLONIC
MYOCLONIC-CLONIC-TONIC
MYOCLONIC -ATONIC
ATONIC
EPILEPTIC SPASMS</td><td>MOTOR
TONIC-CLONIC
EPILEPTIC SPASMS</td></tr>
<tr><td rowspan="2">NON MOTOR ONSET
AUTONOMIC
BEHAVIOUR ARREST
COGNITIVE
EMOTIONAL
SENSORY</td><td rowspan="2">NON MOTOR ONSET
TYPICAL
ATYPICAL
MYOCLONIC
EYELID MYOCLONIA</td><td>NON MOTOR ONSET
BEHAVIOUR ARREST</td></tr>
<tr><td>UNCLASSIFIED</td></tr>
</table>

Seizures are also classified into provoked or unprovoked.[8,9] Seizures caused by immediate brain injury such as secondary to head injury, stroke, neuro-infections such as meningitis or encephalitis, exposure to toxins or drugs, hypoxia, acute metabolic derangements such as hypoglycemia, hypocalcemia, hypo/hypernatremia, hypomagnesemia are called acute symptomatic (also called provoked, reactive, or situational seizures).[8] In children, febrile seizure are also included in this category. The seizures that occur in the absence of the above clinical conditions or beyond the time period estimated for occurrence of symptomatic seizure are termed unprovoked seizures.[9]A seizures,therefore, may be the result from acute brain insult (vide supra) or be the first manifestation of epilepsy.

Acute symptomatic seizures have incidence of 29-39 per 100,000 per year and represent about 40% of all afebrile seizures.[9] In the developing countries, where neurocysticercosis is endemic, acute symptomatic seizures may account for up to 50% cases of new onset seizures.[10] Acute symptomatic seizures in the children are more frequent at younger age. Febrile seizures, head trauma, CNS infections, and metabolic derangements account for majority of the acute symptomatic seizures in children.[8,9,11]

Incidence of new onset unprovoked seizures have varied across studies, ranging from 42.3 to 61.0 per 100,000 per year, with higher incidence in men and children younger than 12 months.[9]In children, incidence of non-febrile seizures has ranged from 89-134 per 100,000 person-years.[12,13] It is estimated that in US, every year, approximately 25,000 to 40,0000 children suffer their first unprovoked seizure.[14]

While there are studies on epilepsy in adolescents,[15-18] studies on new onset seizures in adolescents are sparse. Vast majority of the published studies have included children, adults or mixed cohorts.[19-25] making it difficult to separately compute data for adolescents.

Adolescence is a peculiar time period with unique health issues and neurological disease profile including epilepsy.[26-28] Etiology of acute

symptomatic seizures related to adolescents are likely to be different than those related to childhood and adult onsets.[13] Similarly, epileptic seizure that have onset during adolescence have certain peculiarities that are not seen during childhood or in adults. Also at initial presentation, it is not possible to distinguish between the acute symptomatic seizure or unprovoked seizure. Hence the present study was envisaged to evaluate the profile of new onset seizures in adolescents, and characterize their clinical characteristics and etiological profile.

AIMS AND OBJECTIVES

1 To study clinical characteristics of first onset seizures, both provoked and unprovoked, in adolescents.

2 To determine the etiologies of first onset seizures in adolescents.

3 To study the electroencephalography (EEG) and MR neuroimaging characteristics of first onset seizures in adolescents.

REVIEW OF LITERATURE

New onset seizures in children, adolescents and adults can occur as acute symptomatic seizures (ASS) or can be first manifestation of epileptic predisposition. In the former, seizures result from acute brain insult secondary to trauma, CNS infections, drugs, and toxic or metabolic etiologies.[8] In young children, febrile seizures are also included in the acute symptomatic category. These seizures may account for up to 40% of all new onset seizures,[8] but are not well studied, especially in adolescents. In developing countries, where neurocysticercosis is endemic, acute symptomatic seizures may account for more than 50% cases of new onset seizures.[10] Unprovoked (epileptic) seizures, a large proportion of which is caused by genetic predisposition, may also be result of old brain injury, structural, infectious, metabolic, autoimmune or unknow etiology.[6]

Provoked or Acute Symptomatic Seizures

Huang et al., studied acute symptomatic seizures in Taiwanese children up to 3 years of age. Incidence of acute symptomatic seizures was 0.46 in 100 children. Of the 63 children included in the analysis, 39 were boys and 24 girls. Most common causes of acute symptomatic seizures identified were acute gastroenteritis, encephalitis/ encephalopathy, and bacterial meningitis. This study was limited to young children only, and no adolescents were included.[29]

Rochester study on acute symptomatic seizuresincluded both children and adults. Overall, age-adjusted incidence was 35/100,000 person-years, being higher in men (45/100,000) compared to women 27/100,000). The age-adjusted incidence was highest during the first year of life but was reduced to 15/100,000 in childhood and early adult years. Infections and metabolic seizures accounted for majority of the cases of acute symptomatic seizures during first year of life. From 1-14 years, CNS infections, and head trauma, and from 15-34, withdrawal and head injury were the predominant causes.[21]

In a hospital-based study from Korea, of the 610 children under 15 years of age who were hospitalized due to seizure, 114 (18.7%) belonged to acute symptomatic seizures category, accounted mostly by acute gastroenteritis and metabolic/toxic etiologies.[30] However, only one child was aged beyond 10 years in the cohort. In another study, Lee and Kim from South Korea identified 109 children with acute symptomatic seizures from a cohort of 568 children with convulsive presentation. Acute gastroenteritis was identified as the cause in 33% followed by unspecified encephalopathy in 31%, and metabolic/toxic etiology in 19%. Central nervous system infections accounted for only 11% of the cases.[31]

Chen and colleagues from Taiwan evaluated 319 children under 18 years of age who presented to the emergency department with new onset seizure. Majority (299) children were younger than 6 years. Febrile seizures occurred in 62%. Twenty other children had acute symptomatic etiology including meningitis/meningoencephalitis, electrolyte/metabolic derangements, intracranial hemorrhage, head trauma, and anoxia. Only 10 children in this study were older than 12 years of age.[22]

Study by Idro and colleagues from rural Kenya determined incidence of acute seizures in children aged 0-13 years at 425 per 100,000 per year. More than 80% of the acute seizures were associated with infections with falciparum malaria being the most common (58%) infection associated with acute seizures in children older than 6 months. Other common infections associated with seizure were pyogenic meningitis, respiratory tract infections, and gastroenteritis.[19]

Only few studies on acute symptomatic seizures are available from India but none exclusively studied adolescents.[32-34] Murthy and Yangala analyzed hospital records of patients admitted with seizures over a period of 6 years. Of the total 2531 patients, 526 (22.5%) had acute symptomatic seizures. Neuroinfections including tuberculosis, neurocysticercosis, meningitis etc. accounted for 78% of all the cases,

followed by vascular etiology in 14%. CNS infections such as neurocysticercosis and neurotuberculosis accounted for 95% of children under 16 years of age.[32]

Another study by Narayanan and Murthy reported acute symptomatic seizures in the setting of neurological intensive care unit (NICU). New-onset acute symptomatic seizures accounted for 66 (2.1%) of the 3151 admissions to the NICU over a period of 28 months. Majority (79%) had single seizure, 15% had seizure cluster, and 6% presented withs status epilepticus. Neuroinfections (neurocysticercosis, tuberculoma, meningoencephalitis), and metabolic (hypo-, hyperglycemia, and hyponatremia) etiologies were noted in 21 (32%) each, followed by cerebrovascular events in 14 (21%). This study included patients between 6 months to 80 years. Data on children and adolescents was not provided separately.[33]

Recently, Soni et al., have described their findings on clinical profile and outcome of acute symptomatic seizures in children. Previously healthy children between 3 months to 12 years of age with new onset acute symptomatic seizures were studied. Of the 105 children (71 boys, 34 girls) enrolled 33 were under 12 months of age. Generalised seizures occurred in 67 (64%) and focal in 32 (30%) children. Status epilepticus was observed in 16 (15%). Most common etiology was CNS infections in 86 (82%), accounted largely by meningitis/ meningoencephalitis in 54 (58%), neurotuberculosis in 18 (21%) and neurocysticercosis in 10 (12%). Adolescents were practically excluded from the study as children beyond 12 years of age were not part of the cohort.[34]

Unprovoked Seizures

Unprovoked seizures constitute the true epileptic seizures with lifetime incidence of an afebrile epileptic seizure of 5%. Hauser and colleagues investigated in the incidence of epilepsy and unprovoked seizures in Rochester between 1935-1984. Age-adjusted incidence was 44 per 100,000 person-years for epilepsy and 61 per 100,000 person-years

for all unprovoked seizures. Incidence was high in males and in the first year of life. Overall, distribution of seizure type between generalized and focal seizures was equal but generalized seizures predominated in the first decade. From etiology point of view, age-specific incidence of idiopathic new onset seizures was higher than that of remote symptomatic through first 35 years of life. This study included children as well as adults.[21]

Smith et al from UK retrospectively studied 118 children with first time seizure. Febrile seizures were noted in 75 (64%), acute symptomatic seizures in 13 (11%), and neonatal seizures in 6 (5%). Afebrile epileptic seizures occurred in 24 (17%) children with generalized in 17 and focal in 7. Majority of the children were under 3 years of age, making it difficult to apply these results to adolescents.[35]

In a multicenter, prospective French study (CAROLE; Coordination Active du Reseau Observatoire Longitudinal de 1'Epilepsie), patients aged between 1 month to 95 years of age with new onset seizures were analyzed. Of the 926 individuals with new onset seizures, 542 (59%) were male. Focal seizures were noted in 428 (46%) and generalized in 295 (32%). Seizures remained unclassified in 203 (22%). Idiopathic seizures were observed only in 228 (25%). Cryptogenic and symptomatic etiology was observed in 598% and 18%. Latter group was accounted for by pre- and perinatal insult, cerebrovascular diseases, and head injury. In this study, there were 377 (41%) children between 1 month to 15 years of age, and 155 (17%) were between 15-24 years of age. Age-specific details especially with respect to seizure types and etiology during adolescence were not provided.[23]

King et al. studied 300 consecutive patients (aged 5-83 years) with new-onset unprovoked seizure with no apparent cause. Fifty-nine (20%) children were aged between 5 to 16 years. Focal (n=116) and generalized epilepsy (n=25) was diagnosed in 141 (47%) cases and 159 (53%) remained unclassified on clinical evaluation. After EEG and

neuroimaging, a final diagnosis of epilepsy was achieved in 243 (81%), with generalized epilepsy in 68 (23%), focal epilepsy in 175 (58%), and unclassified in 57 (19%). Most common specific generalized epilepsy syndromes were juvenile absence epilepsy and juvenile myoclonic epilepsy. Temporal lobe epilepsy was the most common focal epilepsy syndrome seen in 57% of the cases of focal epilepsy. This was followed by benign rolandic, and benign occipital epilepsies seen in 7%. First EEG was abnormal in 43%, and with addition of second sleep deprived EEG, 61% of all patients showed epileptiform discharges. Epileptogenic lesions were seen in 38 (14%) of the 277 patient who underwent neuroimaging (MRI in 263, computed tomography alone in 14). Further age-specific details related to adolescent age group were not mentioned in the study.[20]

Shinnar et al., reported results from a prospectively followed cohort of 283 children (aged between 1 month to 19 years) who had first unprovoked afebrile seizure. Study included 159 boys and 124 girls. Mean age at first seizure was 6.7 years. There were 73 children older than 10 years in the cohort. Generalized seizures were noted in 180 (64%) and focal in 103 (36%). Seizures were idiopathic in 236 (83%) and remote symptomatic in 47 (17%). Family history of unprovoked seizures was obtained in 27 (12%) cases. History of prior febrile seizures was present in 42 (15%) cases.[36]

In a community based prospective study from Sweden, Sidenvall et al., computed incidence rate of first unprovoked seizure to be 88 per 100,000 per year in children aged 0-15 years. Generalized seizures were more common than focal seizures. In this cohort of 80 children, 16 were adolescents between 11-16 years of age. In this group also, generalized seizure were more common than focal seizures.[37]

Stroink et al studied 156 children (70 boys) aged 1 month to 16 years with first unprovoked and untreated seizure. Seizures occurred in awake state in 100 (64%) and during sleep in 42 (27%). Based on clinical description, seizures were described as tonic clonic in 142

(91%) and partial (focal) in 14 (9%). Positive family history and previous febrile seizures were noted in 15 (10%) and 17 (11%) children, respectively. Epileptiform abnormalities were noted in 68 (44%) after first EEG and 88 (56%) after 2 EEGs. Neuroimaging abnormalities (computed tomography) were detected in 12 (8%) children. Separate data for adolescents (children > 10 years) was not given.[25]

Vieira et al from Brazil studied 387 children with new-onset seizure. These children were between 68 days to 14.4 years of age and 214 (55%) were male. Seizures were generalized in 167 (43%) and focal in 220 (57%). Interictal epileptiform abnormalities were detected on EEG in 111 (29%) patients only. Neuroimaging abnormalities were detected in 23 % children.[38] Rizvi et al reported their experience from single seizure clinic from Canada. Patients between 14-88 (mean 42.1) years of age were studied. Eighty-two (41%) patients were diagnosed with epilepsy, with generalized epilepsy in 45%, focal epilepsy in 37% and unknown epilepsy in 18%. There were 28 (14%) with single unprovoked seizure. Abnormal EEG was found in 94 % patients with epilepsy.[24]

A recent prospective study of new onset seizures presenting as status epilepticus in children revealed 144 (10%) cases of status epilepticus out of 1,382 infants and children with new-onset seizures. Febrile seizures as the most common cause noted in 46 (32%). Most common acute symptomatic cause was CNS infection. Cerebral dysgenesis was the most common cause in remote symptomatic category. Number of adolescents in this study was not specified.[39]

EEG and Neuroimaging abnormalities

Electroencephalography (EEG) is an important tool for initial investigation in children as well as adults with established or suspected unprovoked new onset seizure. EEG findings are of paramount importance EEG in classifying the seizures and epilepsy. Shinnar and colleagues looked at EEG findings in 347 children with first unprovoked

seizure. EEGs were available in 321 (93%), and 135 (42%) had an abnormal EEG. EEG abnormalities included focal spikes in 57% and generalized spike and wave discharges in 21%. Slowing and nonspecific abnormalities were found in 32% and 0.5% children respectively. Abnormal EEGs were more common in children with remote symptomatic seizures than in those with idiopathic seizures, more common in partial seizures than in generalized seizures and more common in children age >3 years than in younger children.[40] Frequency of neuroimaging abnormalities in the same cohort was found to be 21%. The most common abnormalities were focal encephalomalacia in 16 children and cerebral dysgenesis in 11.[41]

Stroink et al found abnormalities in EEG in 68 (43.5%) out of 156 patients with new onset unprovoked seizures. A second (sleep deprived or during natural sleep) EEG was done if first EEG was unrevealing, and it resulted in identification of epileptiform EEG abnormalities in another 20 children, giving overall yield of 88 (56%). Computed tomography (CT) in 112 patients and was abnormal (mostly atrophy) only in 12 (11%).[25]

Doeschar and colleagues evaluated EEG and neuroimaging data of 356 children with newly diagnosed seizures. Epileptiform EEG abnormalities were noted in 119 (65.7%) with focal abnormalities in 89 (74.8%). Neuroimaging abnormalities were present in 59 (32.6%) patients. A wide variety of abnormalities were identified in these children.[42] Neufeld et al. did a retrospective review of EEG finding in 91 patients (aged above 15 years) with first unprovoked seizures. An abnormal EEGs were obtained in 69% with epileptiform activity in 21%, slowing in 58% and both epileptiform and slowing in 10%. Overall, epileptiform abnormalities were present in 1/3rd (31%) patients.[43]

King et al found EEG abnormalities in 43% after first EEG, and with addition of second sleep deprived EEG, 61% of all patients showed epileptiform discharges. Epileptogenic lesions were seen in 38 (14%) of the 277 patient who underwent neuroimaging (MRI in 263, computed

tomography alone in 14). Majority of the identified lesions were various kind of tumors noted in 17.[20]

Wyman et al., enrolled 73 patients (>17 years of age) with new onset seizure or previous seizure but without EEG. EEG was performed in 71, and 24% of them received a diagnosis of epilepsy. Similar results have been reported by others.[44] Vieira et al., found interictal epileptiform abnormalities on EEG in 29% of the patients; 41 with generalized and 70 with focal abnormalities. In this study, neuroimaging abnormalities were detected in 23 % children.[38] Maytal et al retrospectively reviewed the children presenting to emergency department with new onset seizure and had had computed tomography of the brain. A total of 66 patients (34 boys and 32 girls), aged 1 month to 16 years were included in the study. While 52 (78.8%) patients had normal results, 14 (21.2%) had abnormal results. Patients with focal seizures were more likely to have abnormal CT scan than patients with generalized seizure.[45]

In a community-based study, Berg et al studied 613 children with newly diagnosed epilepsy. Neuroimaging was done in 488 (79.6%) of the 613 children with magnetic resonance imaging in 388 (63%), computed tomography in 197 (32%) and both in 97 (16%). Significant abnormalities were found in 62 (13%), accounted for by tuberous sclerosis in 4, cerebral malformations in 3, tumors in 2, and miscellaneous abnormalities in the remaining.[46] Dirik et al in North Cyprus evaluated magnetic resonance neuroimaging findings in 220 children with newly diagnosed epilepsy. At least 1 MR abnormality was found in 89 (45.45%). Most documented lesions were encephalomalacia, hydrocephaly and brain atrophy (4.5%).[47]

New onset seizures in Adolescents:

Literature describing new onset seizures in during adolescence is scarce. In almost all the studies on new onset seizures, adolescents have been included as part of childhood or adult cohorts. Incidence of new onset seizures from these mixed studies has been found to vary

between 21 to 61 per 100,000 per year.[9] Camfield and colleagues found incidence of epilepsy at 21/100,000 per year for children aged 11-15 years compared with 118/100,000 for children aged <1 year, and 48/100,000 for those aged 6-10 years.[48]

Simard-Tremblay described 65 patients (35 male) with adolescent epilepsy with first seizure between the age of 12-16 years. Family history of epilepsy in first degree relatives was obtained in 7 cases. History of perinatal abnormalities, and prior febrile seizures were noted in 8 and 7 instances respectively. Generalised seizures (58%) outnumber focal seizures (42%). EEG was performed in 62 patients and abnormalities were detected in 53 (85.5%) patients including 46 (74%) with interictal epileptiform discharges. Neuroimaging was abnormal in 9 (15.5%).[18]

Smith and colleagues reported on 173 with adolescent epilepsy. Epilepsy was classified as generalised in 76 (43.9%), focal in 70 (40%) and unclassified in 27 (15.6%). EEG and neuroimaging findings were not described.Few other studies are available but are prevalence studies and have included adolescents with[15,17] but not new onset seizures. Review of literature thus reveals lack of studies devoted to the study of clinical, electroencephalographic, neuroimaging characteristics of new onset seizures exclusively in adolescent children.

MATERIALS AND METHODS

This prospective, hospital based study was conducted in Department of Paediatrics, Dayanand Medical College and Hospital, Ludhiana over a period of 18 months from January 1, 2020 to June 30, 2021. All the adolescents with new onset seizures and attending the paediatric out-patient, in-patient and paediatric emergency services were included in this study. All adolescents with seizures or seizure-like presentation underwent detailed history, physical, and neurological examination identify the relevant cases.

New onset seizure was defined as any episode of seizures occurring for the first time. A cluster of seizures all of which occurring within 24 hours or the occurrence of status epilepticus at onset were regarded as a single seizure. The seizure was considered provoked when there was an immediate identifiable neurological insult such as fever, head trauma, drug/toxin exposure, a metabolic abnormality or meningitis/ encephalitis.[8] The seizure will be considered unprovoked if there was no such identifiable immediate neurological insult.[9]

Following inclusion and exclusion criteria will be employed:

Inclusion Criteria:

- New onset focal or generalized seizure. For unprovoked seizures, adolescents with prior history of neonatal, febrile, or other provoked seizures will not be excluded.
- Adolescents presenting to the department within three months of onset of seizure will be enrolled.
- Age 10 years to 18years
- Gender: either

Exclusion Criteria

- Age < 10 years, > 18 years
- Presentation three months after the onset of seizure
- Parents refusal to participate.

The selected adolescents meeting the inclusion criteria were undergo detailed clinical evaluation by history and physical examination. Laboratory investigations will be dictated by clinical suspicion of the underlying cause of seizures. Similarly, neuroimaging (MRI /CT scan) and electroencephalography (EEG) will be done on the basis of results of clinical evaluation. Details were recorded on a structured proforma. Seizures and epilepsy were classified according to recent ILAE (2017) classification of seizures,[5] and epilepsies.[6]

The patient cohort was analysed for demographic parameters, seizure and epilepsy type and etiological diagnosis. For the purpose of analysis, the cases were divided into two groups. One with acute symptomatic seizures and second with unprovoked seizures, as these two types of seizures differ in etiology, investigations, treatment interventions and prognosis. Statistical analysis was done using SPSS 21.0 version software. The p-values were computed by Chi square test.

OBSERVATIONS AND RESULTS

A total of 51 patients met the inclusion criteria and were included in the study. The demographic and clinical characteristics of these subjects were as:

DEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF SUBJECTS:

Adolescents' age ranged from 10 to 17 years. The mean age of the study group was 2.78 ± 2.08 and median age was 13 (11-15) years. The age distribution of subjects in the study group is depicted in table1. Majority 23 (45%) were between 13-15 years of age, followed by 21(41.1%) between 10-12 years and 7 (13.7%) between 16-17 years. Under the category of Acute symptomatic seizure, majority of 8 (50%) were between 10-12 years of age, followed by 6 (37.5%) between 13-15 years and 2 (12.5%) between 16-17 years. Under the category of Unprovoked seizures, majority 17 (48.6%) were between 13-15 years of age, followed by 13 (37.1%) between 10-12 years and 5 (14.3%) between 16-17 years. There was no statistically significant difference in age distribution between the two groups.

Table 2: Age Distribution of the subjects

						Total	Chi-square value	p-value
		Acute symptomatic seizure		Unprovoked seizure				
AGE	10-12.0	8	50.0%	13	37.1%	21	0.765	0.682
	13-15.0	6	37.5%	17	48.6 %	23		
	16-18.0	2	12.5%	5	14.3 %	7		

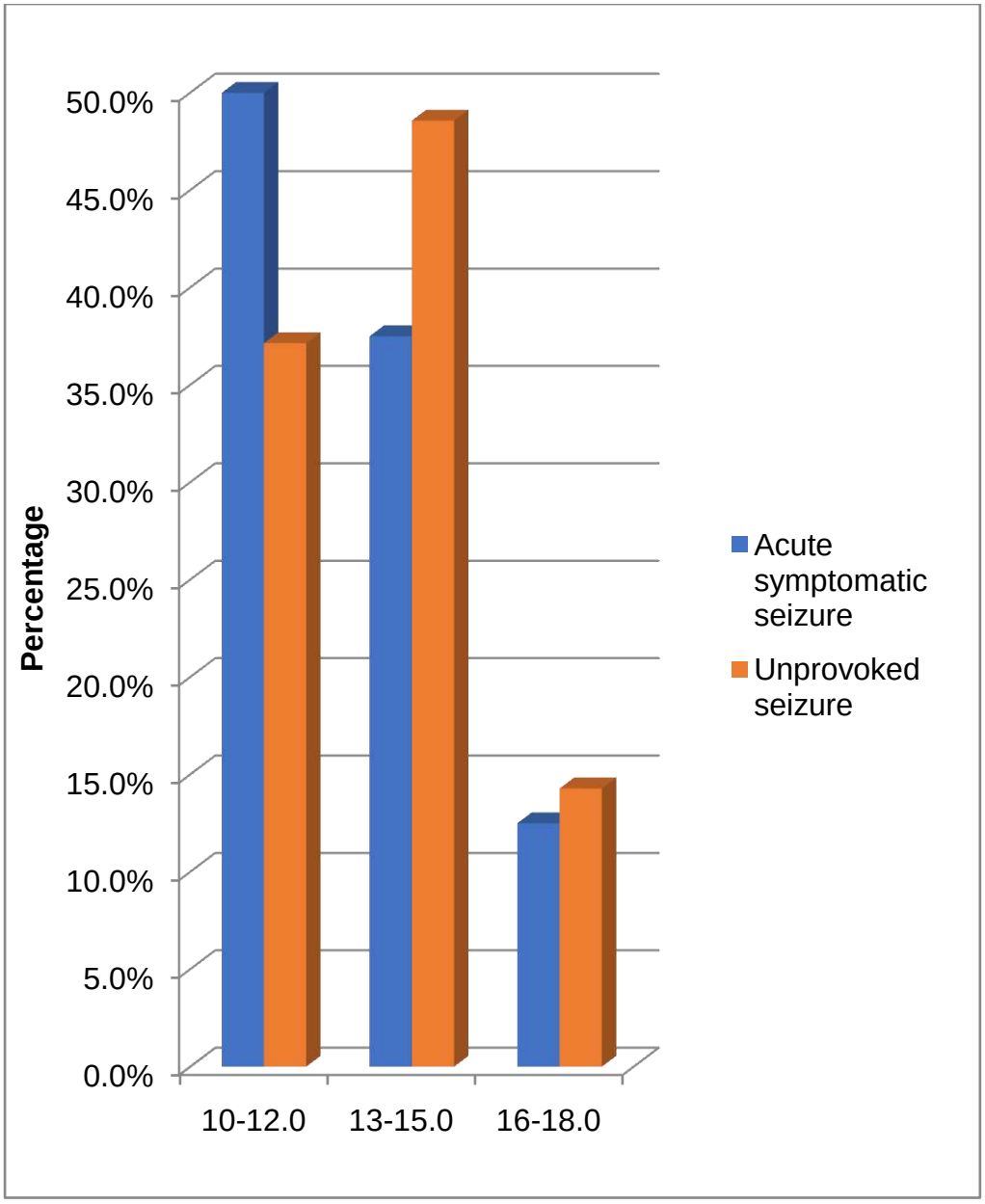

Figure 1: Age Distribution of the subjects

GENDER DISTRIBUTION

As shown in table 3, of the total 51 subjects, 40 (78.4 %) were males and 11 (21.6%) females. With male to female ratio of 3.6:1. The male predominance was noted in both the acute symptomatic seizures, (14 males vs. 2 females) as well as unprovoked seizures (26 males vs. 9 females); however, the observed difference was not statistically significant.

Table 3: Gender distribution of the study group

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
GENDER	F	2	12.5%	9	25.7%	11	1.133	0.150
	M	14	87.5%	26	74.2%	40		
Total		16	100.0%	35	100.0%	51		

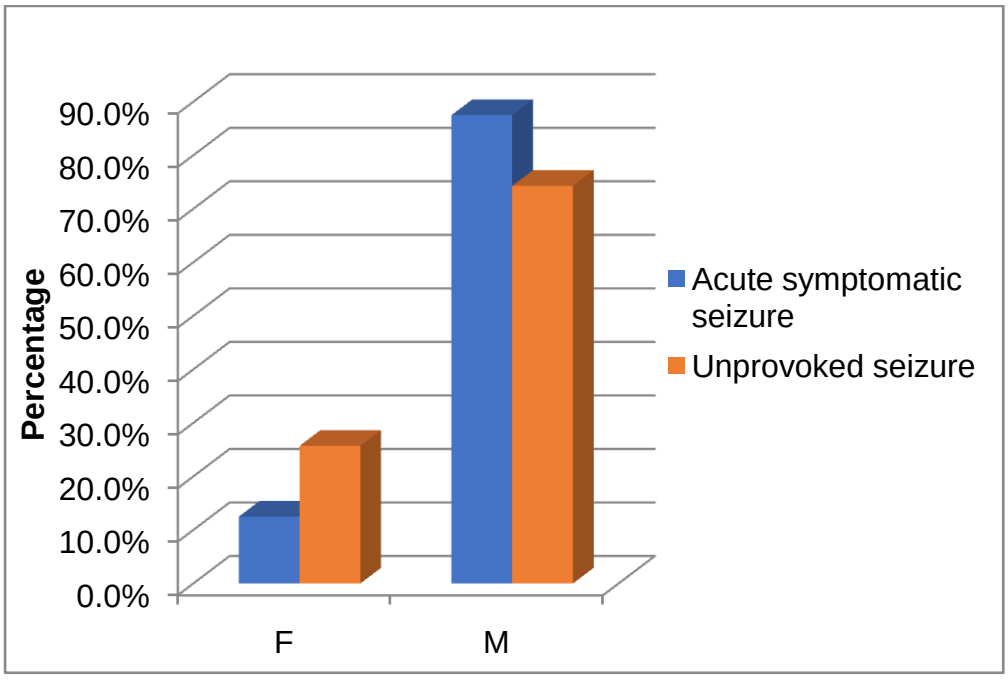

Figure 2: Gender distribution of the study group

AREA OF PRESENTATION

Thirty-five (68.6%) subjects presented to out-patient department (OPD) as compared to 16 (31.4%) who presented to the paediatric emergency department (Table 4). In the ASS group, 11 (68.8%) patients with acute symptomatic seizure presented to the emergency department while 5 (31.3%) presented in OPD. In the unprovoked seizure group, only 5 (14.3%) patients to the emergency department while 30 (85.7%) presented in OPD. Compared with unprovoked seizure group, patients in the ASS group were significantly more likely to present to the emergency department. Similarly, patients in unprovoked seizures group significantly more often presented to the out-patient department when compared with ASS group.

Table 4: Area of presentation

						Total	Chi-square value	p-value
		Acute symptomatic seizure		Unprovoked seizure				
AREA OF PRESENTATION	EMERGENCY	11	68.8%	5	14.3%	16	15.128	0.000
	OPD	5	31.3%	30	85.7%	35		
Total		16	100.0%	35	100.0%	51		

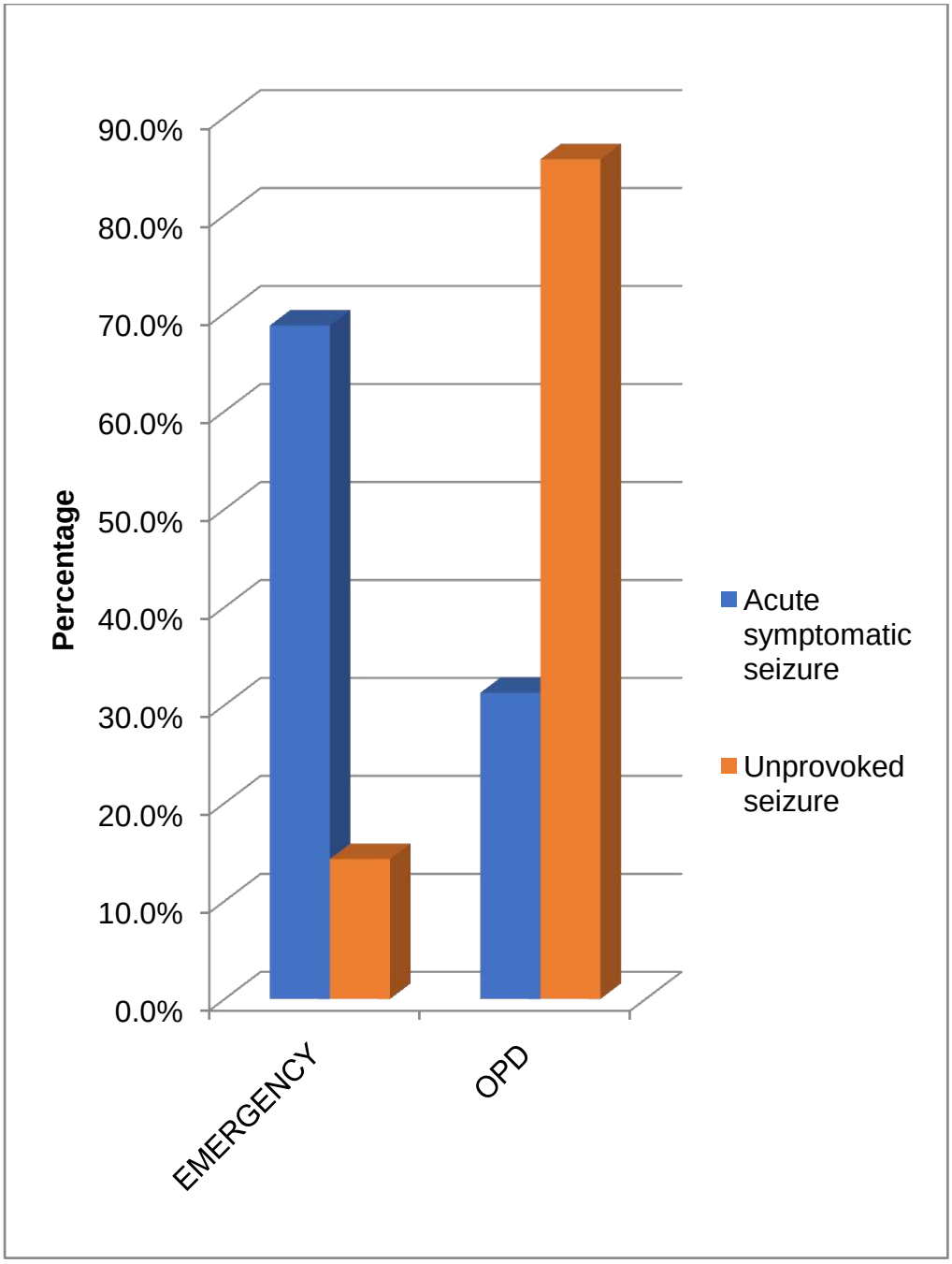

Figure 3: Area of presentation

TIME TO PRESENTATION

Only 3 (5.9%) subjects presented within 24 hours of onset of seizure. Majority 38 (74.5%) were brought to our hospital within 1-7 days of onset of seizure. Table 5 depicts the distribution of cases according to the interval between the onset of seizure and presentation to our hospital. Overall, majority (38 of 51; 74.5%) of the patients were brought to our hospital within 1-7 days after the onset of seizure. While 10 (19.6%) patients were brought after more than 7 days after seizure onset, only 3 patients presented within 24 hours of onset. Time to presentation was however, not statistically significant between the ASS and the unprovoked seizure groups.

Table 5: Time to presentation to hospital after onset of seizure

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
DURATION SINCE SEIZURE	**Within 24hrs**	2	12.5%	1	2.9%	3	1.848	0.396
	1-7 days	11	68.8%	27	77.1%	38		
	> 7 days	3	18.8%	7	20.0%	10		
Total		16	100.0%	35	100.0%	51		

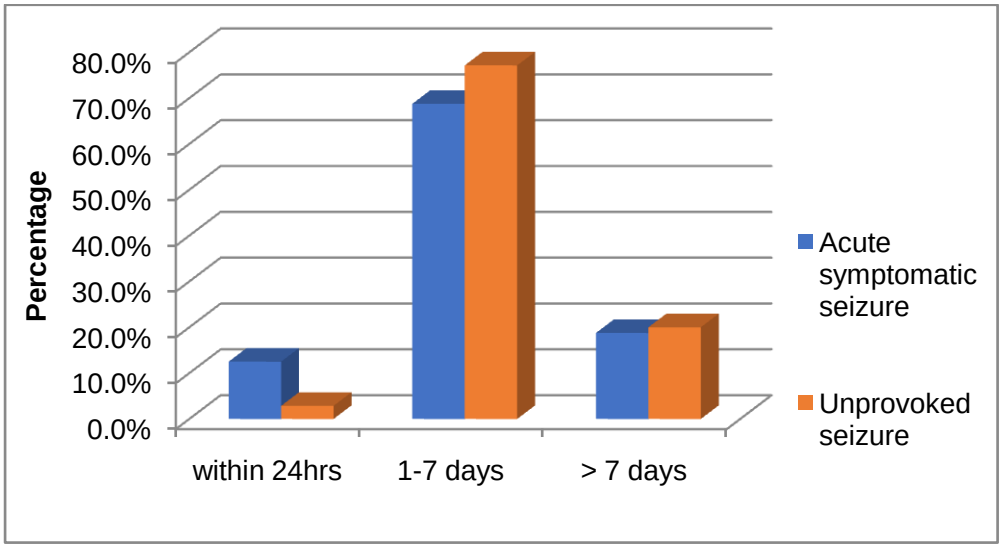

Figure 4: Time to presentation to hospital after onset of seizure

PLACE OF OCCURRENCE

As shown in table 6, overall 42 (82.4%) subjects had their seizure while they were at home whereas 3 (5.9%) children experienced it in the school. Six (11.8%) subjects had seizure in the hospital. Majority of the unprovoked seizure in both the groups occurred at home compared with that occurred outside the home (school, hospital).

Table 6: Place of Occurrence of Seizure

						Total	Chi-square value	p-value
		Acute symptomatic seizure		Unprovoked seizure				
PLACE OF OCCURRENCE	HOME	10	62.5%	32	91.4%	42	8.645	0.013
	HOSPITAL	5	31.3%	1	2.9%	6		
	SCHOOL	1	6.3%	2	5.7%	3		
Total		16	100.0%	35	100.0%	51		

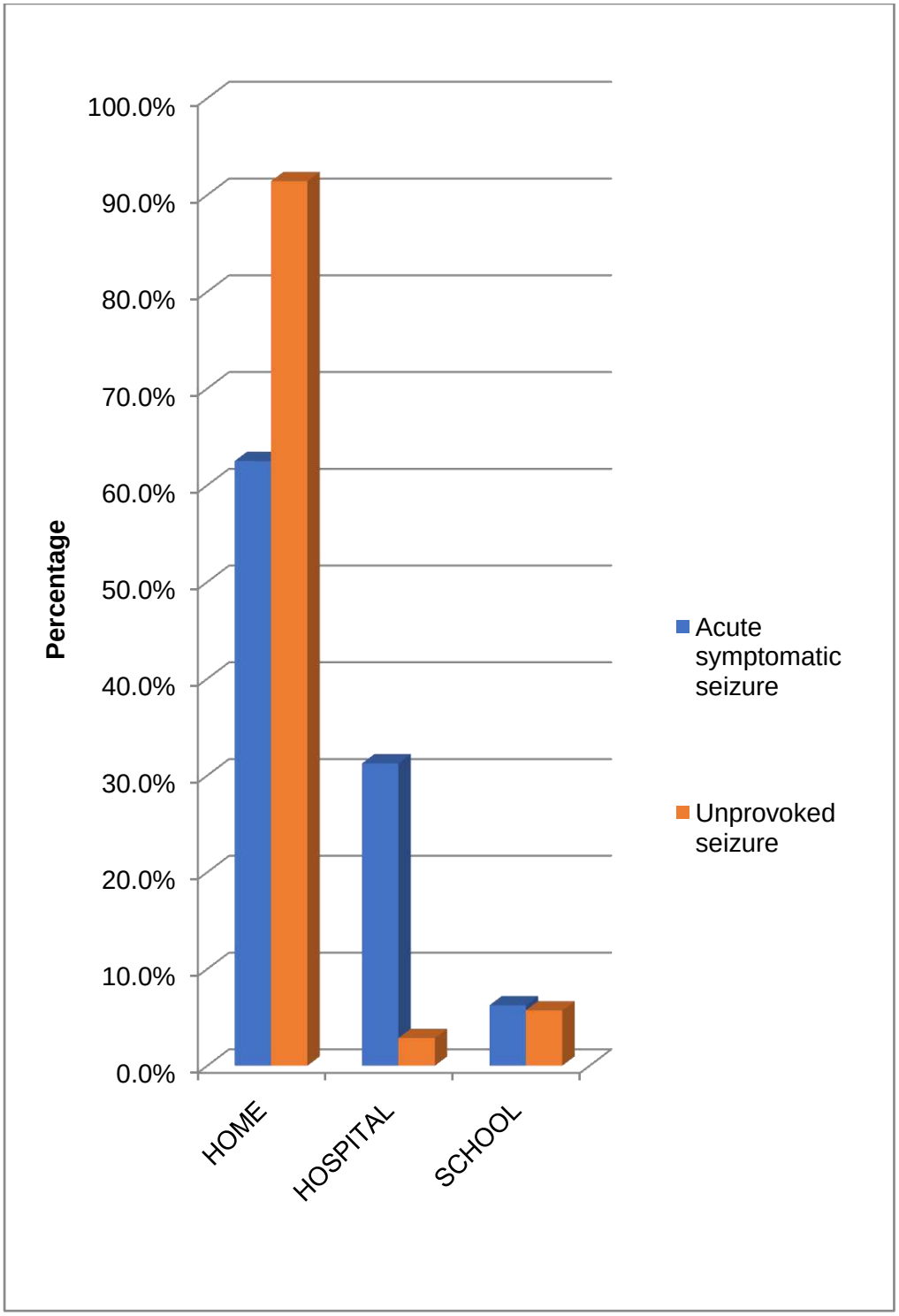

Figure 5: Place of Occurrence of Seizure

CIRCUMSTANCE OF OCCURRENCE OF SEIZURE

More new onset seizures occurred during awake state than during sleep. In all, 29 (56.9%) subjects had their seizure when they were awake, and 22 (43.1%) had it during sleep (Table 7). There was no statistically significant difference in the seizure occurrence during sleep vs. awake period between the ASS and unprovoked seizure groups.

Table 7: Circumstances of occurrence of seizures

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
CIRCUMSTANCE OF OCCURRENCE	AWAKE	8	50.0%	21	60.0%	29	2.281	0.516
	SLEEPING	8	50.0 %	14	40%	22		
Total		16	100.0%	35	100.0%	51		

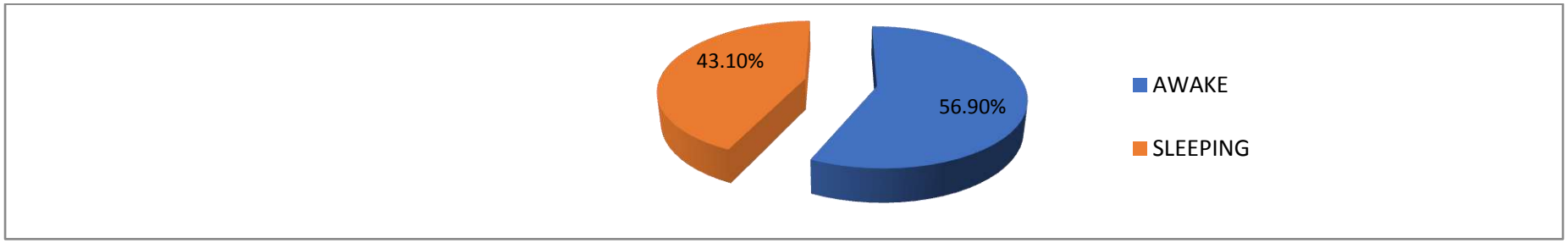

Figure 6: Circumstances of occurrence of seizures

TRIGGERING FACTORS

A (subjective) trigger for occurrence of seizure was identified in 27 (52.9%) subjects and 24 (47.1%) children did not have any identifiable trigger. Seizures occurred while watching screen such as mobile phone /TV (n=10), eating food (n=3), playing (n=7), taking bath (n=3) and studying (n=3). In 8 (50%) patients under acute symptomatic seizure had triggers while 19 (54.3%) had triggers who had unprovoked seizures. The difference between the two groups was not statistically significant.

Table 8: Triggers for new onset seizures

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
Pro-ictus (Triggers)	NO TRIGGERS	8	50.0%	16	45.7%	24	4.848	0.563
	TRIGGERS	8	50.0%	19	54.3%	27		
Total		16	100.0%	35	100.0%	51		

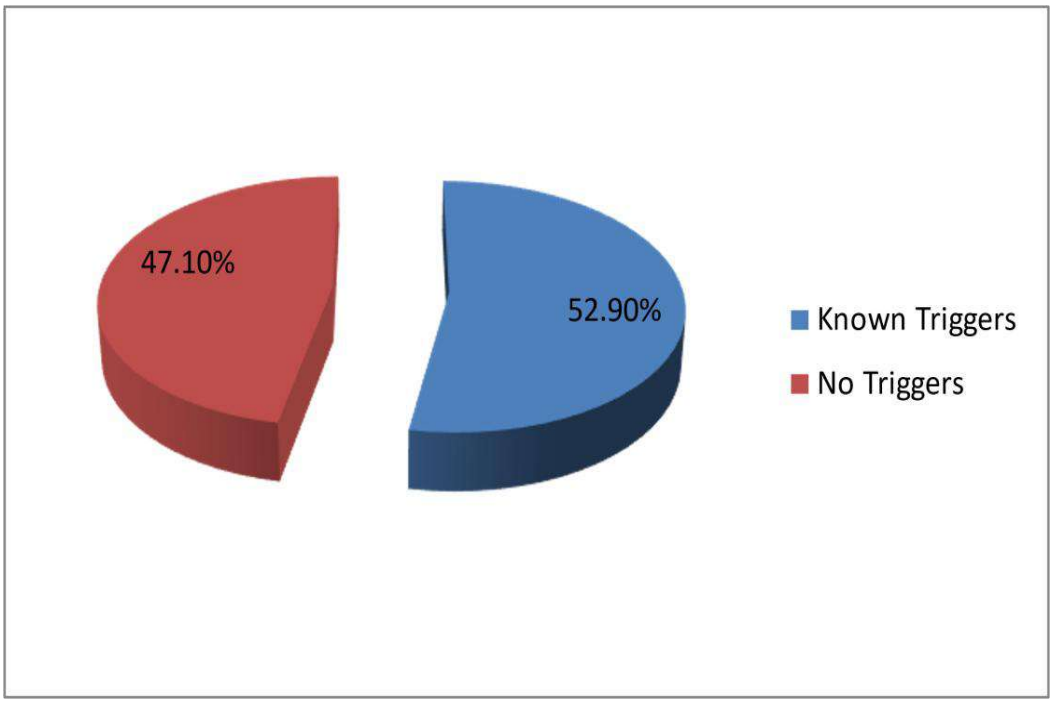

Figure 7: Triggers for new onset seizures

AURA OR WARNING SYMPTOMS

Aura or warning symptoms before the onset of seizure were reported in 16 (31.3%) cases. These warning symptoms were auditory (n=1), gustatory (n=1), vertiginous (n=11), and autonomic (n=1), features. These symptoms are present in 4 (25%) of patients with acute symptomatic seizure while in 12 (34.3%) of patients with unprovoked seizure and the observed difference between the two groups was not statistically significant.

Table 9: Warning symptoms (Aura) before seizure onset

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
Pre-ictus (Aura)	PRESENT	4	25.0%	12	34.3 %	16	6.942	0.225
	ABSENT	12	75.0%	23	65.7%	35		
Total		16	100.0%	35	100.0%	51		

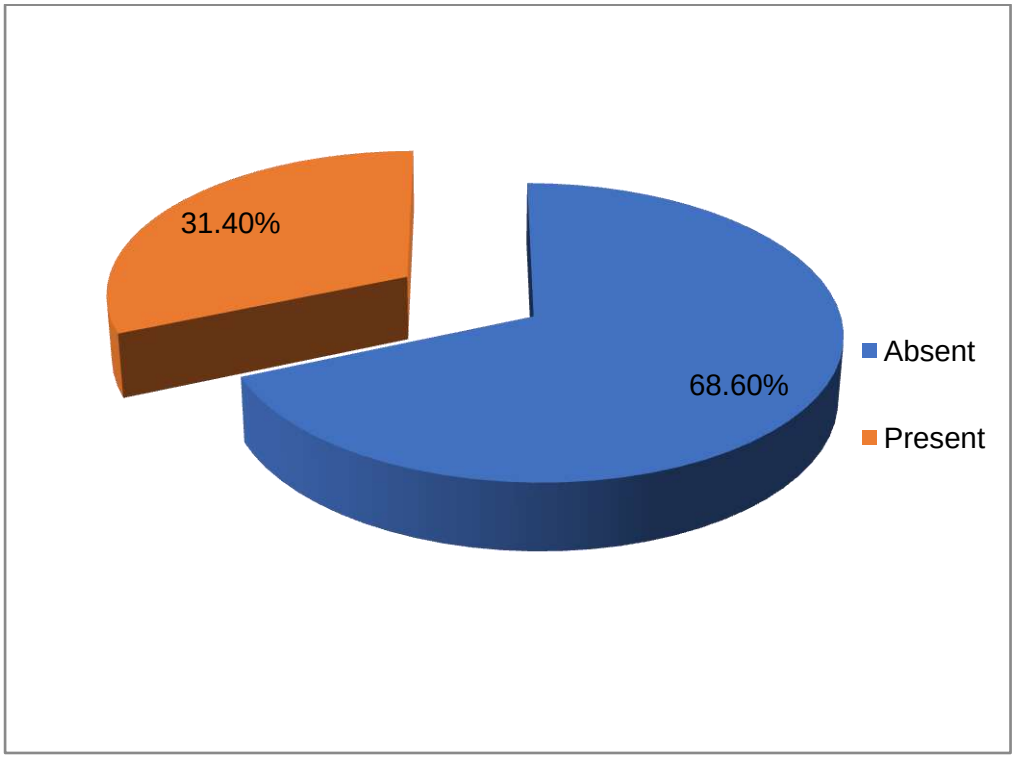

Figure 8: Warning symptoms (Aura) before seizure onset

ONSET OF SEIZURE EPISODE

Generalized-onset seizures were the predominant type, noted in 32 (62.7%) patients. Focal-onset seizure was noted in 15 adolescents (29.4%). In 4 (7.8%) children, seizure onset was unclassified. The distribution of focal and generalised onset seizures between the ASS and unprovoked seizure groups was not statistically significant.

Table 10: Types of Seizure

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
SEIZURE TYPE	FOCAL	4	25.0%	10	28.5%	14	0.572	0.903
	Generalized	11	68.8%	22	62.8%	33		
	Unclassified	1	6.3%	3	8.6%	4		
Total		16	100.0%	35	100.0%	51		

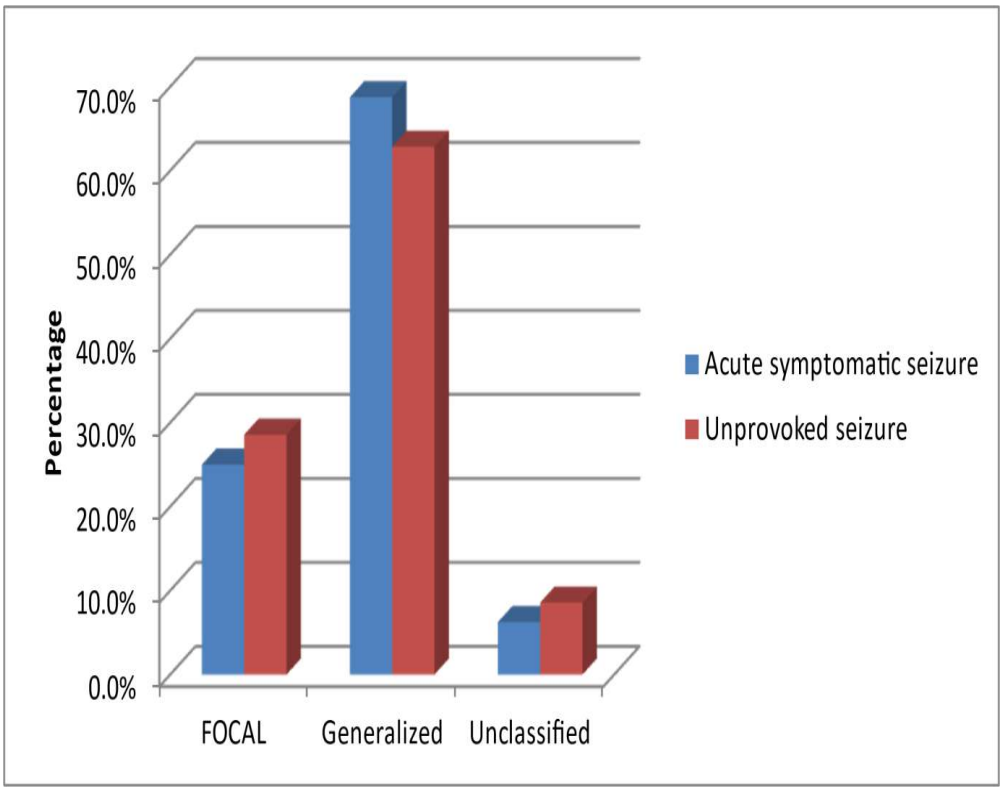

Figure 9: Types of Seizure

FOCAL ONSET: LEVEL OF AWARENESS

Of the 15 patients with focal onset seizures, awareness was impaired in 11 (73.3%) whereas awareness was preserved in 4 (27%). When the ASS and unprovoked seizure groups were compared for this parameter, there was no statistically significant difference between the groups

Table 11: Focal seizure according to level of awareness

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi- square value	p- value
	AWARE	2	66.6%	2	18.8 %	4 (28.5)	1.471	0.689
AWARENESS	IMPAIRED AWARENESS	1	33.3%	9	81.8 %	10 (71.4)		
	Total	3	100.0%	11	100.0%	14		

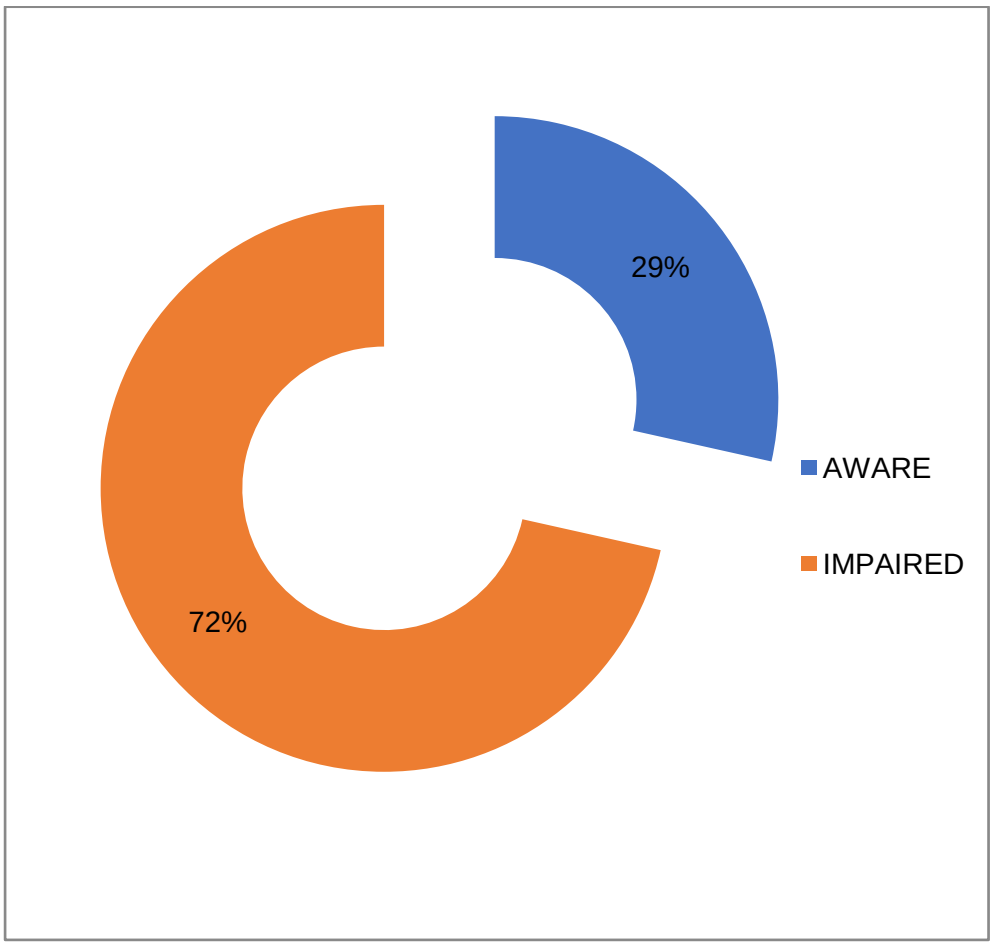

Figure 10: Focal seizure according to level of awareness

MOTOR AND NON-MOTOR SEIZURES

Motor seizures dominated the presentation both in focal and generalised onset seizures. All the 33 adolescents with generalised seizures were motor type and none had non-motor presentation. Of the 14 adolescents with focal onset seizures, 11 (78.5%) had motor and 3 (21.4%) had non-motor manifestations (Table 12).

Table 12: Distribution of motor and non-motor seizures

Seizure Type	Sub- Types	Focal seizures	Generalised seizures
Motor	Tonic clonic	-	23 (69.6%)
	Clonic	6 (42.8%)	1 (3%)
	Myoclonic-tonic-clonic	-	1 (3%)
	Tonic	5 (35.7%)	5 (15.15%)
	Myoclonus	-	3 (9%)
Non-motor		3 (21.4%)	0
Total		14	33

POST ICTAL PHASE

Post ictal phase was described in 32 (62.7%) subjects. Symptoms like headache, abnormal behaviour, amnesia, confusion, sleepiness, vomiting, and unconsciousness has been seen in post ictal phase. There was no difference between the ASS group and the unprovoked seizure group as far as occurrence of post ictal symptoms is concerned (Table 13).

Table 13: Post- ictal symptoms

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
Post-ictal	PRESENT	9	62.5%	22	65.7%	32	13.224	0.585
	NONE	7	37.5%	12	34.3%	19		
Total		16	100.0%	35	100.0%	51		

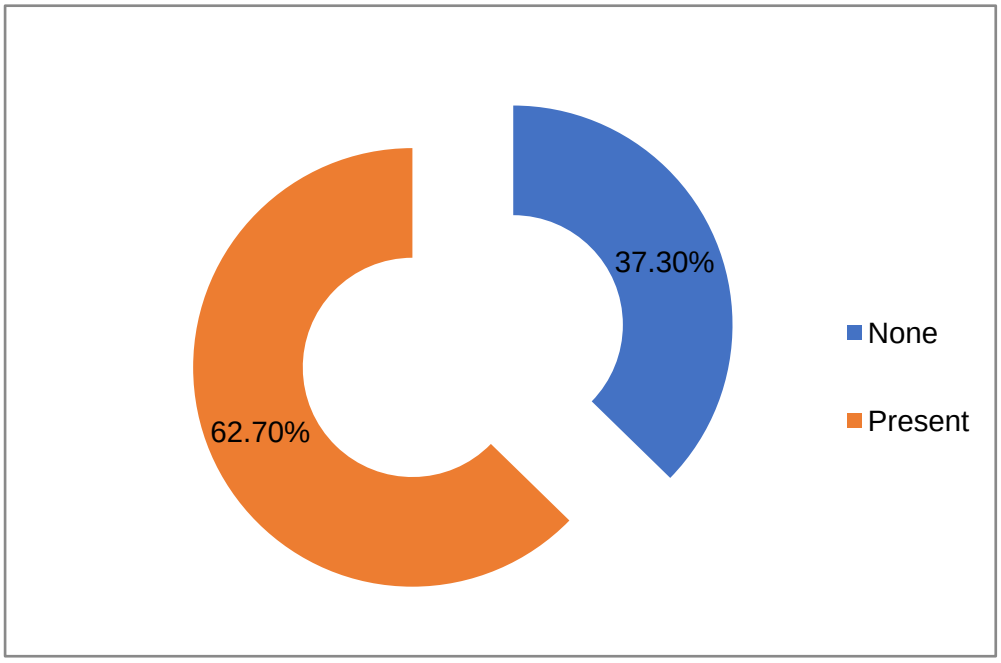

Figure 11: Post- ictal symptoms

TOTAL DURATION OF SEIZURE

As shown in table 14, of all the children included in this study, 9 (17.6 %) had seizures lasting less than 5 minutes, 22 (43.1%) between 5 to 30 minute and 20 (39.2%) more than 30 minutes. Presentation with status epilepticus (seizure > 30 minutes) was observed in 4 (25%) of ASS group and 16 (45.8%) of unprovoked group. When duration of seizure was compared between the groups, there was no difference between the ASS group and the unprovoked seizure group.

Table 14: Duration of seizure between the two seizure groups

						Total	Chi-square value	p-value
		Acute symptomatic seizure		Unprovoked seizure				
Duration	LESS THAN 5 MIN	4	25 %	5	14.2%	9	6.221	0.183
	5-30 MIN	8	50 %	14	40%	22		
	>30 MIN	4	25 %	16	45.8%	20		
Total		16	100.0%	35	100.0%	51		

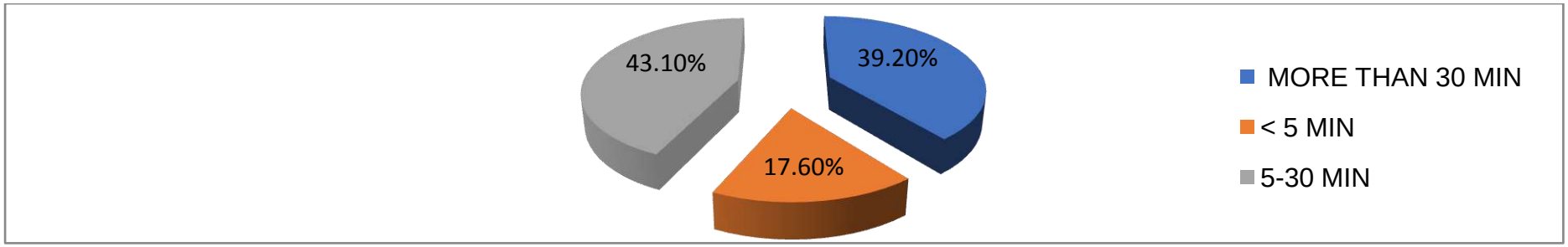

Figure 12: Duration of seizure between the two seizure groups

CLUSTERING: PRESENT/ABSENT

As shown in table 15, clustering of seizures was seen in 11(21.6%) of 51 subjects, including 4 (25%) in the acute symptomatic group and 7 (20%) in unprovoked seizure group. No statistically significant difference was observed between the groups.

Table 15: Occurrence of seizure clusters

						Total	Chi-square value	p-value
		Acute symptomatic seizure		Unprovoked seizure				
Clustering of seizures	ABSENT	12	75.0%	28	80.0%	40	0.162	0.687
	PRESENT	4	25.0%	7	20.0%	11		
Total		16	100.0%	35	100.0%	51		

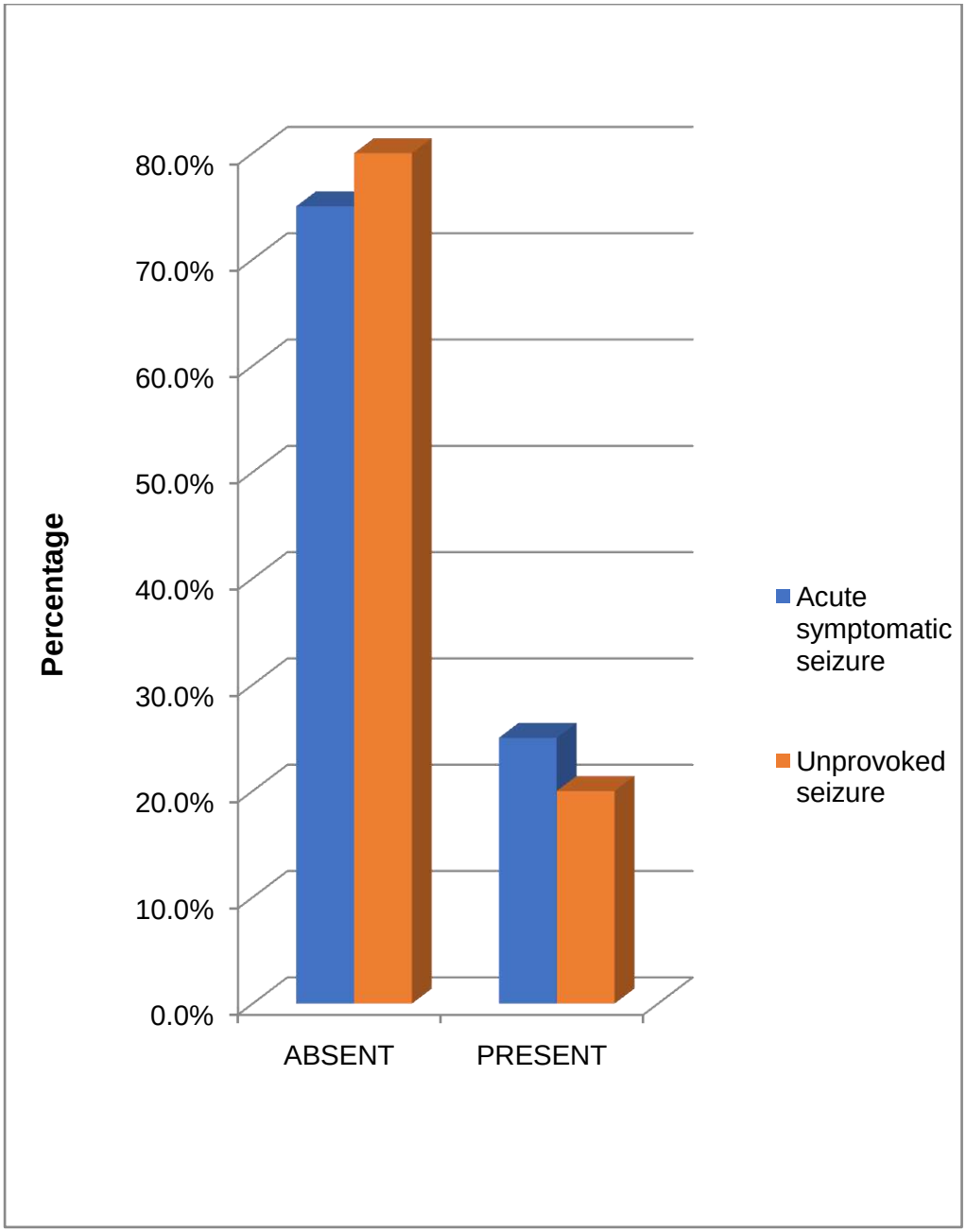

Figure 13: Occurrence of seizure clusters

PATIENT HAVING MORE THAN ONE SEIZURE TYPE

All except 1 patient had only one type of seizure at presentation.

Table 16: Patients having more than 1 seizure types

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
More than one kind of seizures	N	16	100.0%	34	97.1%	50	0.466	0.495
	Y	0	0.0%	1	2.9%	1		
Total		16	100.0%	35	100.0%	51		

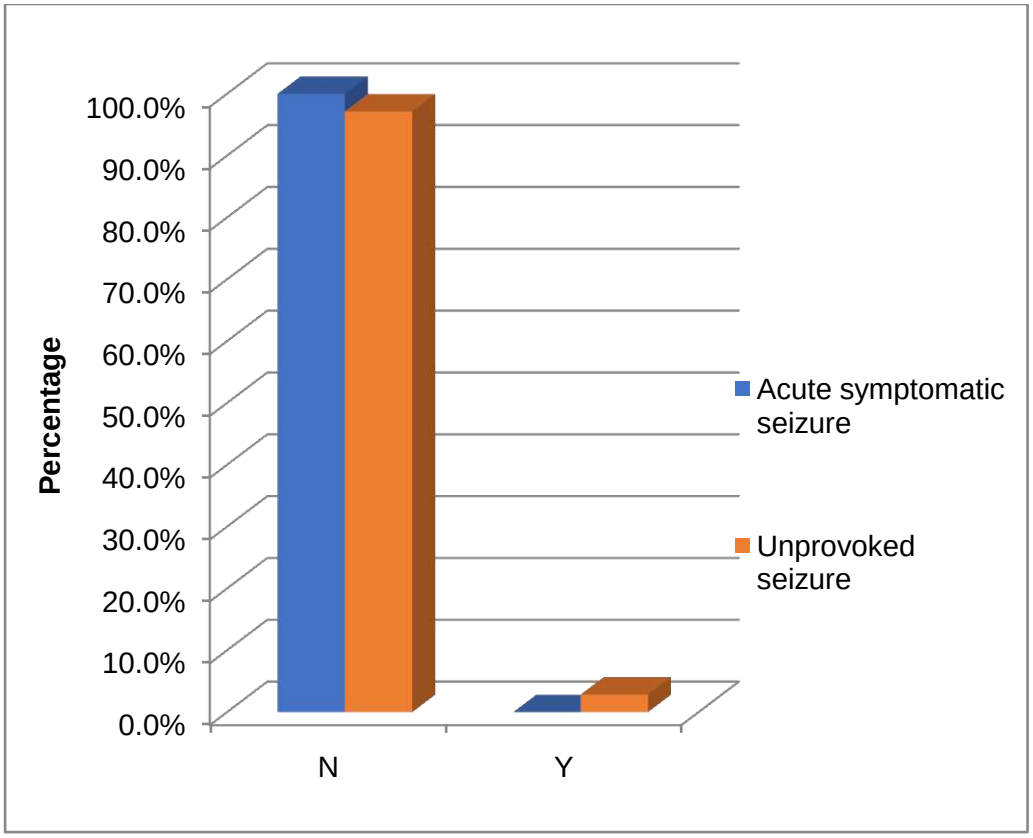

Figure 14: Patients having more than 1 seizure types

FAMILY HISTORY

Family history of epilepsy or seizures was noted in 5 children, with 4 children belonging to unprovoked seizure group, and 1 child belonging to ASS group. When family history was compared between the groups, there was no statistically significant difference between the two seizure groups.

Table 17: Family history of seizure or epilepsy

						Total	Chi-square value	p-value
		Acute symptomatic seizure		Unprovoked seizure				
Family History of seizures	NONE	15	93.8%	31	88.6%	46	0.333	0.564
	PRESENT	1	6.3%	4	11.4%	5		
Total		16	100.0%	35	100.0%	51		

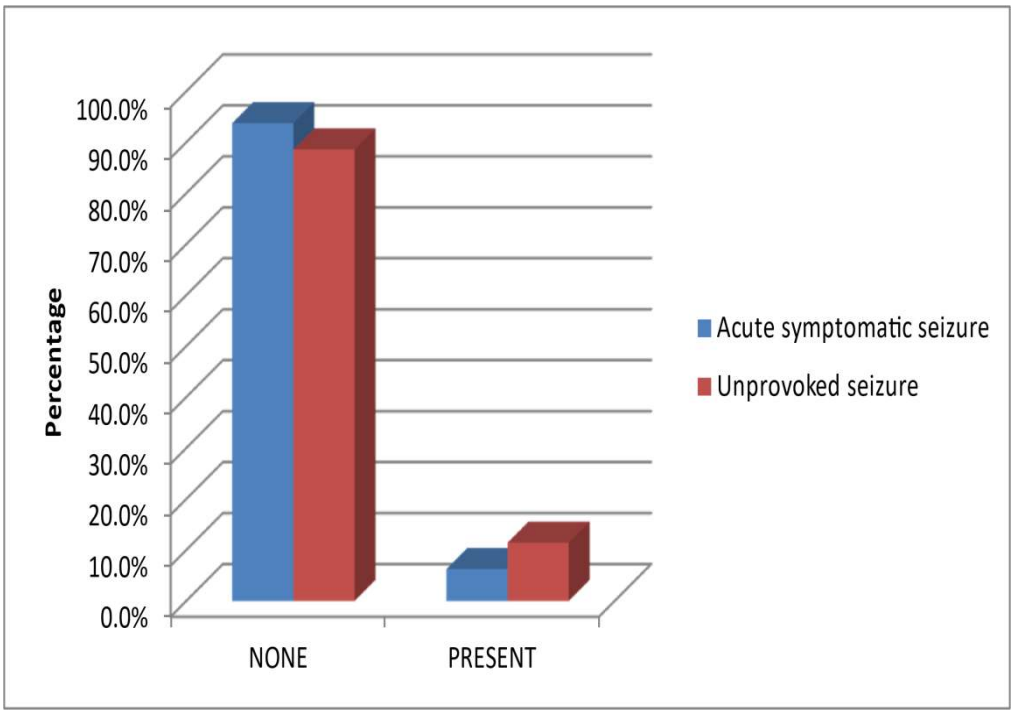

Figure 15: Family history of seizure or epilepsy

NEUROLOGICAL EXAMINATION

Six (11%) patients had abnormal neurological examination on initial presentation (table 18). All 6 patients belonged to ASS group. None in the unprovoked group had abnormal neurological findings.

Table 18: Neurological examination findings

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
Neurological Exam	ABNORMAL	6	37.5%	0	0.0%	6	14.875	0.001
	NORMAL	10	62.5%	35	100.0%	45		
Total		16	100.0%	35	100.0%	51		

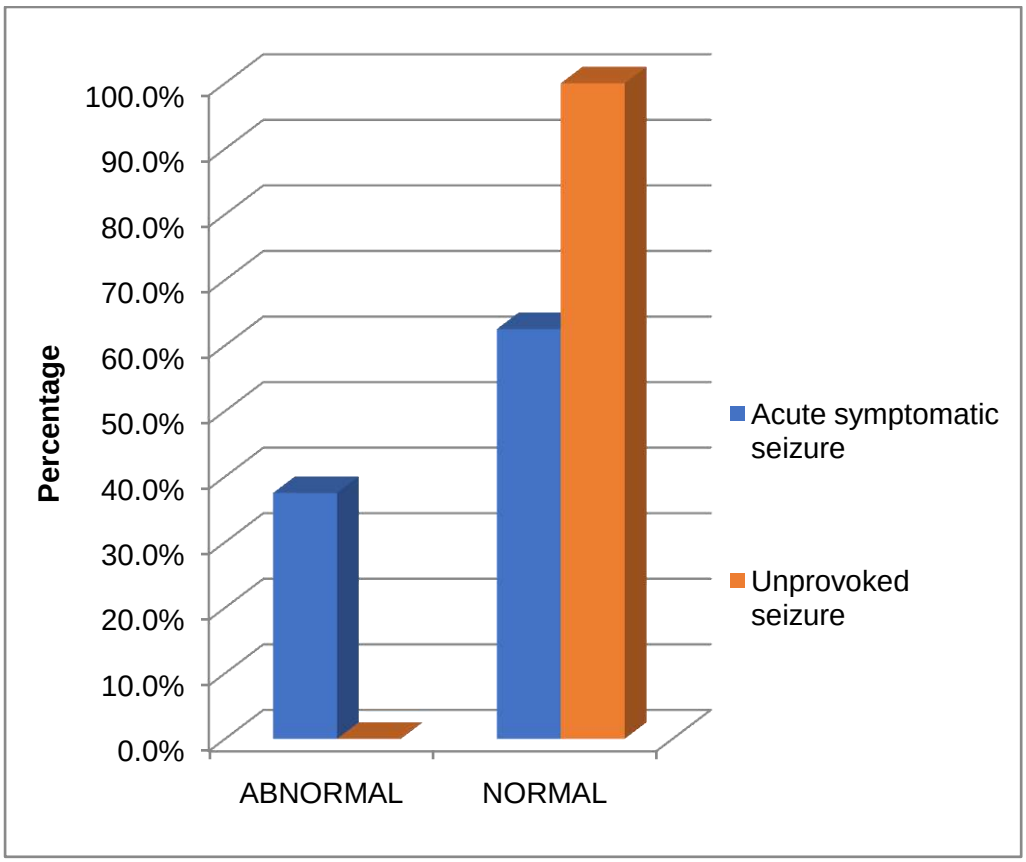

Figure 16: Neurological examinations findings

EEG

EEG was done in 38 (74.5%) children. In the unprovoked seizure group, 32 (91.4%) of 35 children, and in the ASS group in 6 (37.5%) of 16 underwent an EEG. None of the children in the ASS group had abnormal EEG. In the unprovoked seizure group, 14 (43.2 %) of the 32 children who underwent EEG had abnormal findings. Abnormal EEG findings included focal spikes in 8 children and 5 had generalised spike and wave discharges, and 1 child had both focal and generalised discharges.

TABLE 19: EEG findings

						Total	Chi-square value	p-value
		Acute symptomatic seizure		Unprovoked seizure				
EEG	ABNORMAL	0	0.0%	14	40.0%	14	19.381	0.000
	NOT DONE	10	62.5%	3	8.6%	13		
	NORMAL	6	37.5%	18	51.4%	24		
Total		16	100.0%	35	100.0%	51		

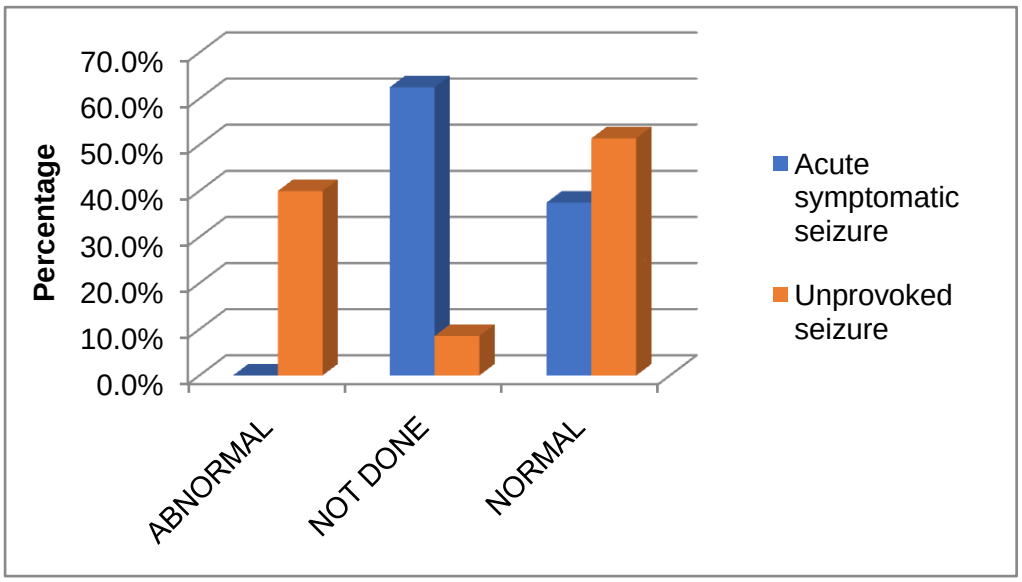

Figure 17: EEG findings

Neuroimaging Findings:

Overall 47 (92.1%) children underwent a neuroimaging study with head CT in 15 (29.4%) and brain MRI brain in 41 (80.3%). Thirty-two children had only brain MRI and 6 only head CT as neuroimaging abnormality. Nine children had neuroimaging with both CT and MRI.

In all 18 (38.2%) children showed neuroimaging abnormalities; 15 on MRI and 3 on head CT.

CT Findings

Computed tomography (CT) findings: In our study, of the 15 children (8 in the ASS group and 7 in the unprovoked seizure group) who had head CT, 7 (46.6%) showed abnormalities, and all the 7 belonged to ASS group. Of the 6 children who only had head CT, 3 showed an abnormality. Abnormal head CT was significantly more common in the ASS group compared with the unprovoked seizure group (Table 20) Abnormal head CT findings included neurocysticercosis in 3, post-traumatic intracranial haemorrhages in 4.

Table 20: Head CT findings

						Total	Chi-square value	p-value
		Acute symptomatic seizure		Unprovoked seizure				
HEAD CT	ABNORMAL	7	43.8%	0	0.0%	7	18.036	0.000
	NOT DONE	8	50.0%	28	80.0%	36		
	NORMAL	1	6.3%	7	20.0%	8		
Total		16	100.0%	35	100.0%	51		

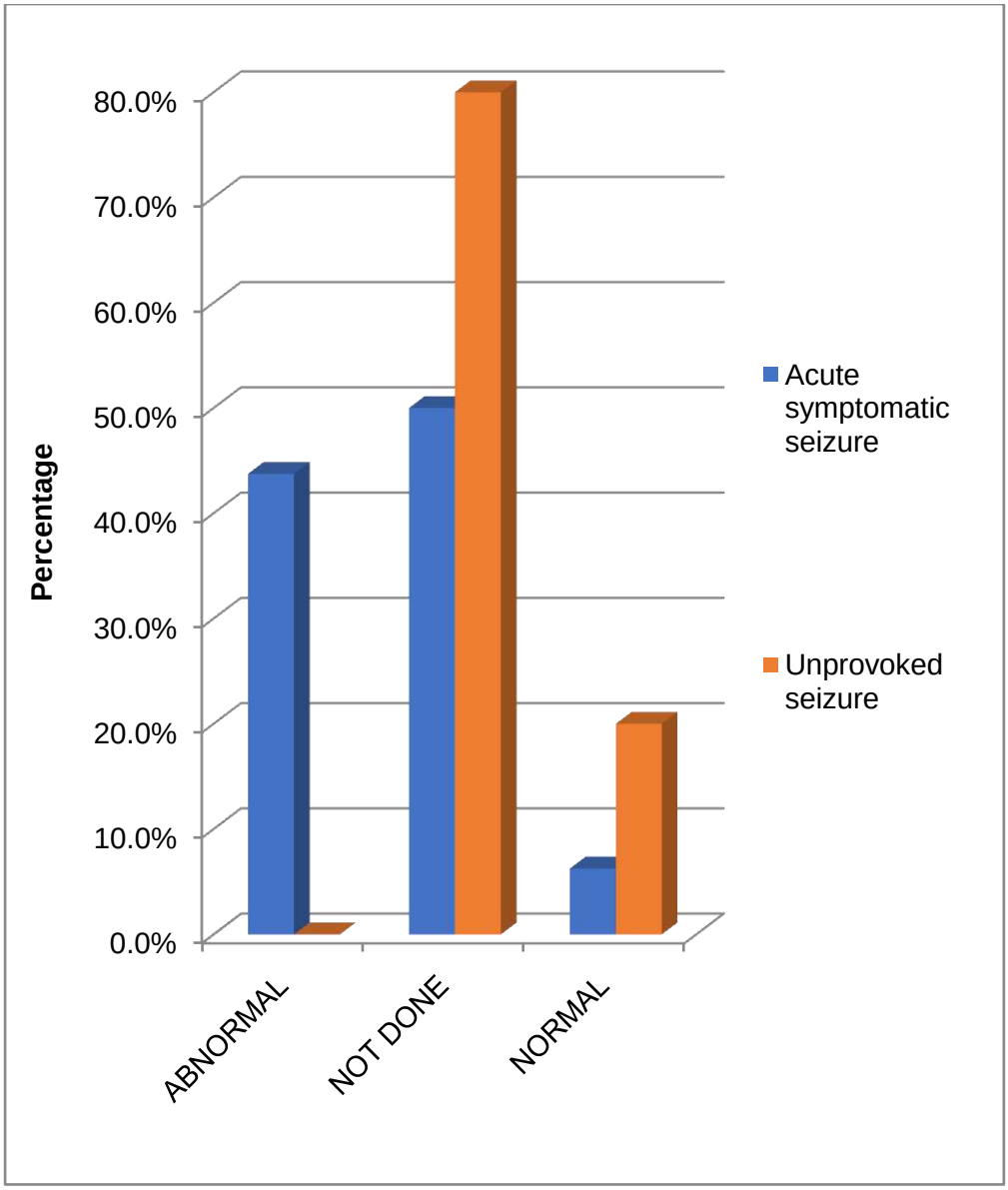

Figure 18: Head CT findings

Brain MRI

Brain MRI was done in 41 children including 11 in the ASS group and 30 in the US group (Table 21). Abnormal findings were observed in 15 (29.4 %) patients with 9 (56.3%) in the ASS group and 6 (17.14 %) in the US group. The ASS group was significantly more likely to display abnormal MR neuroimaging compared to the US group (p=0.007). The individual abnormalities on MRI were consistent with neurocysticercosis in 3, gliosis/encephalomalacia in 3, meningitis in 2, encephalitis in 2, traumatic intracranial bleed in 1 and hydrocephalus in 1. Remaining 3 children had non-specific hyperintensities in the cerebral (n=2) and cerebellar white matter (n=1).

Table 21: Brain MRI findings

		Acute symptomatic seizure		Unprovoked seizure		Total	Chi-square value	p-value
BRAIN MRI	ABNORMAL	9	56.3%	6	17.14%	15	14.009	0.000
	NOT DONE	5	31.3%	5	14.2%	10		
	NORMAL	2	12.5%	24	68.5%	26		
Total		16	100.0%	35	100.0%	51		

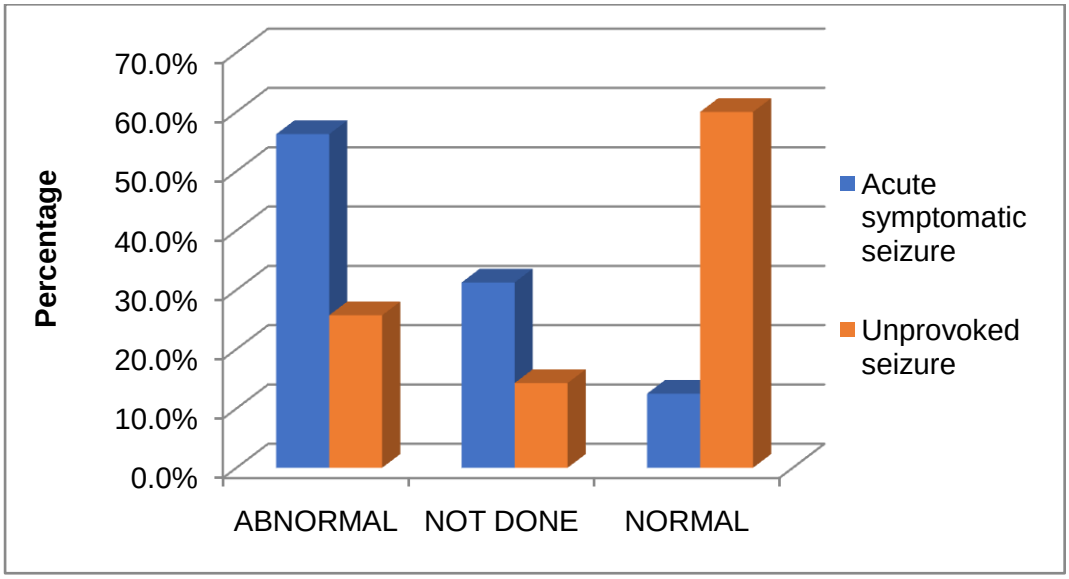

Figure 19: Brain MRI findings

Final Diagnosis

Final etiological diagnosis of the new onset seizure in the ASS group and the US group is given in the Table 22 and Table 23. In the ASS group, CNS infections accounted for 8 (50%) cases followed by head injury in 4 and metabolic etiology in 4 (hypocalcemia due to vitamin D deficiency in 2 and due to hypoparathyroidism in 2). Of the 8 with CNS infections, neurocysticercosis accounted for 4 (50%).

Acute Symptomatic Seizure

Table 22- Etiological diagnosis in acute symptomatic seizure

Acute symptomatic seizure	No. of cases (% age)
Neuro-infection	8 (50)
Neurocysticercosis	4 (25)
Encephalitis	2 (12.5)
Meningitis	1 (6.25)
Tuberculous meningitis	1 (6.25)
Hypocalcemia	4 (25)
Vitamin-D deficiency	2 (12.5)
Hypoparathyroidism	2 (12.5)
Head Injury	4 (25)
Total	16

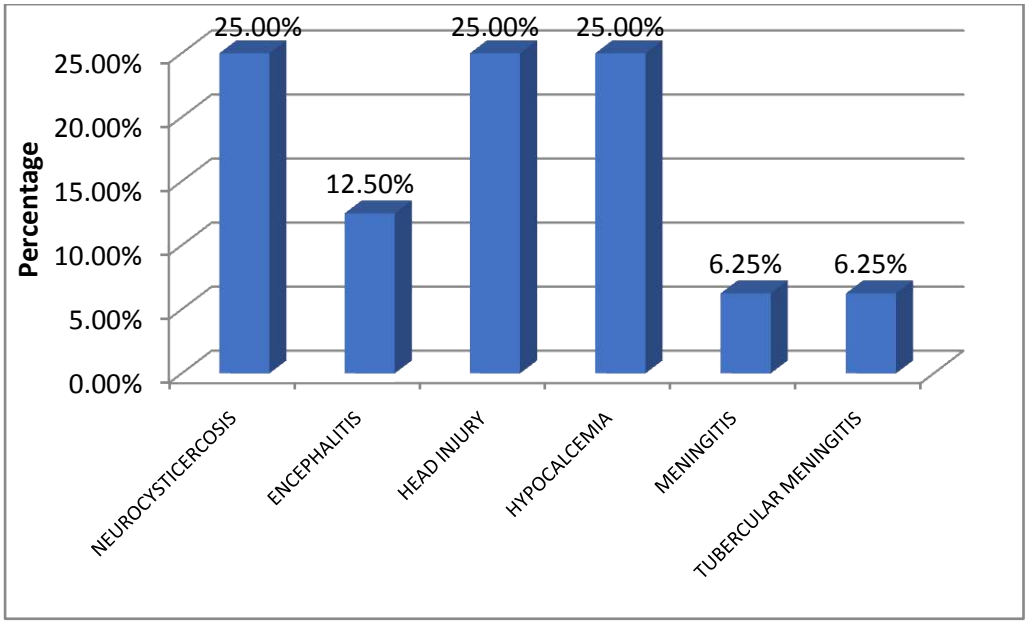

Figure 20- Etiological diagnosis in acute symptomatic seizure

UNPROVOKED SEIZURE

New-onset unprovoked epileptic seizures were genetic or presumed genetic in origin in 23 (65.7%). Four children had structural etiology,. The etiology remained unknown in 8 (22.8%) (Table 23).

Table 23: Final diagnosis in unprovoked seizure group

Final Diagnosis in Unprovoked seizure	No. of cases	Percentage
GENETIC	23	65.7%
STRUCTURAL	4	11.4 %
UNKNOWN	8	22.8 %
Total	35	100.0%

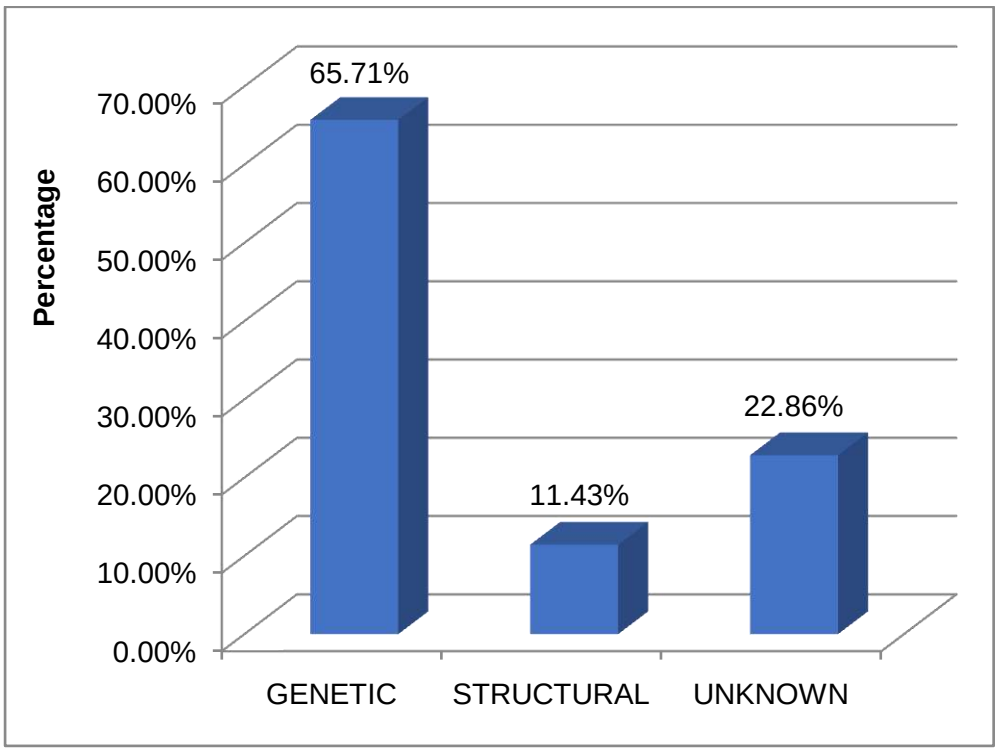

Figure 21: Final diagnosis in unprovoked seizure group

DISCUSSION

New onset seizures in adolescents, like at any other age, can be caused by an acute brain insult (called acute symptomatic seizures), or be a presenting symptom of epileptic predisposition (called unprovoked or epileptic seizures).[8,9] While there are several studies evaluating new onset seizures in children and adults, none have specifically studied adolescents. In most of these studies, adolescents have been the part of the mixed cohorts that have included children and/or adult.[10-13, 19-25] To fill this gap in knowledge and to understand the demographics and clinical characteristics of new onset seizures in adolescents, we undertook the present study.

During an 18-month period, between January 2019 and June 2020, we recruited 51 patients aged 10-18 years with new onset seizures irrespective of whether caused by immediate brain insult or were the result of epileptic predisposition. Reasons were twofold: at presentation, often it is not possible to distinguish between the two categories of seizures on clinical grounds only without resorting to laboratory (such as metabolic profile) and radiological (such as neuroimaging) investigations.[49] This distinction between the two types of seizures is important to the definition of epilepsy and to the prognosis.[9] Secondly, we wanted to find out relative proportion of acute symptomatic seizures in the new onset seizures and also to determine its etiological spectrum.

It has been estimated that acute symptomatic seizures may accounts for 40% of total seizures in all ages and up to 50% in countries where neurocysticercosis is endemic.[9,50,51]Acute symptomatic seizures occurred in 16 (31.4%) of the 51 adolescents with new onset seizures, thus accounting for almost 1/3rd of all the new onset seizures in the cohort. We believe, our enrolment of patients was significantly affected by concomitant Covid (SARS-CoV-2) pandemic. Access to health care facilities including non-covid emergencies was severely curtailed during the pandemic.[52,53]

Male patients have been affected more frequently than females in previous studies.[21,23,29,34-36] We also observed male preponderance inn our study but was quite marked compared with other studies. To some extent it can be explained on the basis of gender bias (in seeking medical attention) that is quiet prevalent in our country.

In our study, 35 (68.6%) patients presented to paediatric out-patient department, while only 16 (31.4%) attended emergency services. In the acute symptomatic seizure group, 11/16 (68.7%) presented to emergency services compared with 5/35 (14.3%) in the unprovoked seizure group. Corresponding figures for presentation to out-patient department for these two groups of new-onset seizure patients are 31.3% and 85.7%. These differences between the acute symptomatic seizure and unprovoked seizure groups were statistically significant. Obviously, children with acute symptomatic seizures are more sick, and frequently have additional symptoms and signs of acute brain insult prompting presentation to emergency services. The current SARS-CoV-2 pandemic possibly affected the way patients with medical emergencies including seizures availed health care facilities.[52,53]

Only 12.5% of the acute symptomatic seizure group and 3% of the unprovoked seizure group patients came to our hospital within 24 hours of occurrence of seizure. Majority (69% of the acute symptomatic seizure group and 77% of the unprovoked seizure group) of new-onset seizure patients presented to our facility between 1-7 days after the occurrence of seizure. One reason for late presentation in the setting of medical emergency could be the current SARS-CoV-2 pandemic which seriously affected the access to medical facilities. Secondly, our hospital is a tertiary care centre, and many patients might have presented to the local health care facility before they were referred.

We also studied the place of occurrence of new-onset seizure. To the best of our knowledge, this has not been reported before. Interestingly, in our study 42 children including 10 in acute symptomatic seizure group and 32 in unprovoked seizure group had their first seizure while

they were at home. Only 3 children were at school when they had their first seizure. Six children (5 in the acute symptomatic group) had their first seizure in the hospital. One reason could be that children including adolescents spend a great deal of their daily time at home, and only about up to 8 hours daily in school. Our study coincided with the occurrence of SARS-CoV-2 pandemic which resulted in school closure and restriction of out-door activities for several months. As a result, most of the time children stayed in-doors.

Epileptic seizures are known to have close relation with circadian rhythm of sleep-wake cycle, and occurrence of seizures during sleep is well known with certain types of epileptic seizures.[54] In our study, 29 (57%) children had their seizure during wakefulness and 22 (43%) during sleep. There was statistically no significant difference between the acute symptomatic seizures and unprovoked seizures. This distinction between sleep and wake seizures may not be significant factor for acute symptomatic seizures, but has great importance in the diagnosis of epilepsy and epileptic syndromes.[12] Occurrence of new-onset seizure during sleep is also a risk factor for recurrence.[55] In the CAROLE study by Jallon et al, new-onset seizure occurred during wakefulness in 75% and during sleep in 24%. This study included patients between the age 1 month to 95 years.[23] Also epilepsies like benign occipital and benign rolandic epilepsies which predominantly occur during sleep have onset in childhood and tend to remit in adolescence reducing the proportion of adolescents who had seizures during sleep.

Activities like watching TV, mobile or other screens are known to precipitate seizure in predisposed individual. Other activities such as eating (eating epilepsy), reading (reading epilepsy), bathing (hot-water epilepsy) etc., can trigger epileptic seizures in predisposed individuals.[56] In our study, these activities were recorded in significant number of adolescents with new-onset seizures. Association of these ‘triggers’ with causation of seizure may not be established on first

seizure. Consistent occurrence of these factors with repeated seizures helps identify these factor as definitive triggers. In some cases, these triggers can be used as a provocation method during EEG to establish their causal association with epileptic seizures. Since we studied the new onset seizures, the occurrence of these triggers could possibly be described as chance occurrence, unless seizure recurrence in future with same trigger is observed.[56]

Aura or warning symptoms are encountered in focal epilepsies and its occurrence suggests focal-onset seizure. Aura was noted in 12 (34%) children with unprovoked seizures. Both acute symptomatic and unprovoked seizure patients reported aura, and there was no significant difference in aura occurrence in these groups. Occurrence (or lack of it) of aura, therefore cannot differentiate between acute symptomatic and unprovoked seizures. Aura symptoms, of course, have lateralization and localization value in focal-onset seizure.

In our study, we have employed the recent ILAE 2017 classification of seizures and epilepsies to new onset unprovoked seizures. Overall, generalised-onset seizures occurred in 32 (62.7%) patients 32 and focal-onset seizure in 15 (29.4%). Of the unprovoked epileptic seizures, seizure onset was generalised in 60%, focal in 31% and unknown in 9%. This is similar to study by Smith et al from UK.[35] In this study, afebrile epileptic seizures occurred in 24 (17%) children with generalized in 17 (71%) and focal in 7 (29%). This study was limited to children, majority of whom were 3 year old or younger. Shinnar and colleagues found generalized seizures in 180 (64%) and focal in 103 (36%).[36] Jallon et al., studied new-onset seizures in children and young adults between 9-39 years of age. Focal seizures were more common and noted in 428 (46%) while generalized seizures occurred in 295 (32%). Seizures remained unclassified in 203 (22%).[23] King et al. studied 300 consecutive patients (aged 5-83 years) with new-onset unprovoked seizure with no apparent cause. Focal epilepsy was diagnosed in 38.6%, generalized in 8.3%, and 159 (53%) remained

unclassified on clinical evaluation. Only 59 (20%) subjects were aged between 5 to 16 years in this study.[20]Hauser and colleagues have reported equal frequency of generalised and focal seizures in first unprovoked seizures.[21] Discrepant results between the studies are likely due epidemiological, demographic and methodological factors.

A few studies have applied ILAE classification to the retrospective cohorts of epilepsy and found focal epilepsy to be more common than generalised epilepsy. Egesa and colleagues retrospectively applied ILAE 2017 classification to cohort of children and adults with epilepsy. Focal onset epilepsy was diagnosed in 62%, generalised in 33% and unknown onset in 5%.[57] Murthy et al., reported focal-onset epilepsy in 43%, generalised in 23% and unknown onset in 33% children aged between 5-15 years.[58] Another retrospective study on children aged between 1-16 years found focal onset epilepsy in 48%, generalised onset in 28%, and unknown onset in 20%.[59] All these studies differed from our study being in retrospective in nature, including patients with established epilepsy diagnosis and also not being limited exclusively to adolescents.

Further classification of focal and generalised seizures in our cohort revealed that motor seizures outnumbered non-motor seizures and the tonic-clonic seizure was the most frequent motor seizure type (Table 12). In the focal seizures, focal unaware seizures (82%) were more frequent than focal aware (18%) (Table 11). Focal motor were more common than non-motor type. Similar to our results, Suvasini and colleagues found focal aware seizures only in 6.8% and focal unaware in 93.2%. Focal motor were more frequent than non-motor. In this study, generalised motor seizures were noted in 68% and non-motor in 32%. In the motor category, myoclonic and tonic-clonic seizures were more common, and in the non-motor category, absences were the most common.[59] Unlike our study which included children aged between 10-18 years, this study included children of all ages between 1 to 16 years.

EEG findings have been reported in variable number of patients with new onset seizures. In our study, we considered only epileptiform abnormalities in EEG to be significant and relevant to the diagnosis. Epileptiform abnormalities were observed in 14 (32%) of the 32 patients, with focal epileptiform abnormalities in (57%) and generalized abnormalities in 29%. This is similar to study by Shinnar et al., who found epileptiform EEG abnormality in 105/321 (32%). Of these, 73% had focal and 27% had generalised spikes.[40] Similar EEG findings were reported by Doeschar et al.,[42] Neufeld et al,[43] and Wayman et al.[44]

We found neuroimaging abnormalities in 18 (32%) adolescents with new onset seizures. These results are in agreement with those reported by others. Kings et al., found epileptogenic lesions in 14% cases,[20] Berg et al., in 13%,[46] and Doeschar et al. 32.6%.[42] Dirik et al., in North Cyprus evaluated magnetic resonance neuroimaging findings in 220 children with newly diagnosed epilepsy. At least 1 MR abnormality was found in 89 (45.45%). Most documented lesions were encephalomalacia, hydrocephaly, and brain atrophy (4.5%).[47] The spectrum of neuroimaging abnormality however has varied widely between these studies, and may be due to methodological and geographic variations in the studies. Most importantly, unlike our study which was exclusively limited to children, other studies have included only children and adults or mixed populations of children and adults.

From the etiology point of view, CNS infections accounted for majority (50%) of the acute symptomatic seizures. Neurocysticercosis was the single most common cause of new-onset of seizures, and occurred in 25%.Neurocysticercosis is endemic to our country and accounts for between 8.7 to 50% of new onset seizures.[10,50,51] Seizures due to NCC have been categorised under acute symptomatic seizures and under unprovoked seizures in various studies making comparison difficult.[60, 61] We have included our patients under acute symptomatic seizures category because acute seizures in patients with NCC are

caused by inflammatory edema, and resolution of NCC occurs within few months of onset, obviating the need for long-term anti-epileptic drugs. Only in small proportion of patients, NCC becomes the cause of chronic epilepsy. Other CNS infections causing acute symptomatic seizures in our study included bacterial/viral meningitis, encephalitis, and tuberculous meningitis. In Rochester study of new onset acute symptomatic seizures, CNS infections, and head trauma were the most common cause of seizure from 1-14 years of age and withdrawal and head injury were the predominant causes and from 15-34 years of age.[21] Similar to our study, Murthy and Yangala found CNS infections such as neurocysticercosis and neuro-tuberculosis in 95% of children under 16 years of age.[32]

We also encountered 4 children with hypocalcemia, secondary to hypoparathyroidism, and vitamin D deficiency. We routinely screen adolescents with new-onset seizures for hypocalcemia to avoid missing a treatable cause of seizures and also to avoid misdiagnosis of epilepsy. Vitamin D-deficiency is especially common during adolescence especially in the developing countries,[62] and can cause neurological manifestations including seizures. [63-65]

In the unprovoked seizures, genetic or presumed genetic etiology of new-onset seizures was the most common etiology, followed by structural causes. Cause remained unknown in 23%. Of the structural lesions, gliosis/encephalomalacia due to neonatal hypoglycemic brain injury, ischemic injury, and hydrocephalus were encountered. In the study by Hauser et al, idiopathic (as per old ILAE seizure classification) new-onset seizures accounted for 65% of cases.[21] Suvasini et al., applied ILAE classification to retrospective cohort of children with epilepsy, and found etiology in 75% cases. Perinatal etiology was the most frequent, and 25% had unknown etiology.[59]This study included children of all ages and adolescents formed only 23% of all cases. Egesa and colleagues found infectious and structural etiology in 45% and 36% cases, and 25% had unknown etiology.[57] This study included children and young adults.

There are limitations to our study. The study was conducted between January 2020 to June 2021and the study period coincided with that of Covid (SARS-CoV-2) pandemic. The pandemic affected several aspects of interaction between the health care seeker and health care providers. Out-patient, inn-patient as well as emergency services for non-covid patients were affected. This resulted in fewer patients visiting our centre, thereby reducing total number of patients recruited for this study. Even access to laboratory investigations such as MRI and EEG was limited during this period. In children who had normal awake EEG, we could not do sleep deprived EEG.

In summary, our study is first attempt to study new-onset seizures exclusively in adolescents, and we included both acute symptomatic and unprovoked seizures. Acute provoked seizures are majorly caused by CNS infections, head injury and metabolic derangements. Unprovoked seizures are largely due to genetic or presumed genetic causes followed by structural causes. Further studies including larger number of adolescents are needed to confirm our findings.

SUMMARY AND CONCLUSION

In conclusion, new onset seizures in adolescents have their own peculairities and characteristics different than those of children and adults. Boys were more frequently affected than girls. Generalised seizures were more common than focal onset seizures. Of the seizure subtypes, motor seizures were more frequent than non-motor seizures with generalised tonic clonic seizures being the most common generalised motor seizures. Of the focal-onset seizures, focal unaware seizures were the most common. EEG and neuroimaging were the two most helpful tools in determining the etiology of seizures. Acute symptomatic seizures account for about 1/3rd of all new onset seizures, and are largely caused by central nervous system infections, traumatic brain injury and metabolic abnormalities. Of the CNS infections, neurocysticercosis was the most common, followed by meningitis, and encephalitis. Metabolic derangements were due to hypocalcemia. Unprovoked seizures occurred in 2/3rd adolescents and were largely caused by genetic or presumed genetic etiology, followed by structural etiology. In a significant number of adolescents etiology of new onset seizures remained unknown. Our study is first of its type to study clinical and etiological characteristics of new-onset seizures in adolescents. Further studies are needed in larger cohorts to corroborate our findings.

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PROFORMA

S.NO-

Name Age/Gender Date of Birth

MRD No. Place of presentation: OPD/IPD/ Emergency

Referring Diagnosis:

Chief complaint:

Narrator:

Duration:

Settings (Para-ictal):

Place of occurrence of seizure: Home/School/Others

Seizure witnessed by:

Circumstances of occurrence:

Awake/On Awakening/Drowsy/Sleeping

Time of the day

If in sleep, time since asleep:

If awake, what was child doing:

Signature of candidate :

Signature of consultant:

Pro-ictus (Triggers):

TV/videogames/working on computer/Mobile

Sleep deprivation/Alcohol/Drugs/Exams/

Pre-ictus (Aura):

None/Visual/auditory/sensory/olfactory/gustatory/vertiginous/psychic/autonomic

Describe:

Duration:

Per-Ictus:

Generalized onset

Motor: Tonic-clonic/Clonic/Tonic/Myoclonic/Myoclonic-tonic-clonic/

Myoclonic-atonic/Atonic/epileptic spasms

Non-motor:

Absences: Typical/atypical/myoclonic/eyelid myoclonia

Focal onset:

Aware/Impaired awareness

Motor: Automatism/Atonic/clonic/epileptic spasms/hyperkinetic/

myoclonic/tonic

Non-motor: Autonomic/behavior arrest/cognitive/emotional/sensory

Signature of candidate : **Signature of consultant:**

Focal to bilateral tonic clonic

Unknown onset:

Motor: tonic clonic/epileptic spasms

Non-motor: behavioral arrest

Unclassified

Duration:

Post-ictal: None/Confusion/amnesia/headache/sleepiness/vomiting/psychosis/

/abnormal behaviour/Todd's paralysis

Duration:

Total duration of seizure:

Clustering of seizures

Present/absent

Describe

Does the patient have more than one kind of seizures? No Yes (specify)

Has someone video-recorded the seizure episodes?

What was done at the time of seizure?

Signature of candidate :

Signature of consultant:

Any additional information if any

Review of systems:

Family History

Past Medical History

Medications

Birth history: Prematurity/HIE/neonatal jaundice/neonatal hypoglycemia/Meningitis/ encephalitis/Stroke/others

Development: Normal/Intellectual disability/Autism/others

Examination Wt Ht

General Physical Exam

Dysmorphism

Neurocutaneous signs

Systemic Exam

Neurological Exam

EEG

Signature of candidate : **Signature of consultant:**

CT/MRI

Others:

Final Diagnosis:

Acute symptomatic seizure: Etiology

Unprovoked seizure: Genetic/Structural/Infectious/metabolic/Immune/Unknown

Signature of candidate :

Signature of consultant:

KEY TO MASTER CHART

D	Day
F	Female
M	Male
MIN	Minutes
N	No
ND	Not Done
WK	Week
WNL	Within Normal Limits
Y	Yes

Sr. No	NAME	AGE	GENDER	DOB	AREA OF PRESENTATION	CHIEF COMPLAINT	NARRATOR	DURATION SINCE SEIZURE	PLACE OF OCCURRENCE	SEIZURE WITNESSED BY	CIRCUMSTANCE OF OCCURRENCE	Time of the day IF IN SLEEP,TIME SINCE SLEEP	Time of the day If awake, what was child doing	Pro-ictus (Triggers)
1	KS	10	M	11/21/2009	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	9 D	HOME	PARENTS	SLEEPING	<5 MINUTES		
2	GS	13	M	3/17/2008	EMERGENCY	ABNORMAL BODY MOVEMENTS	FATHER	10 D	HOSPITAL	FATHER	SLEEPING	<5 MINUTES		
3	A	15	M	12/19/2004	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	4 D	SCHOOL	TEACHER	AWAKE		PLAYING	PLAYING
4	DK	10	F	1/12/2010	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	1 D	HOME	MOTHER	AWAKE		WATCHING MOVIE	WATCHING MOVIE
5	GS	14	M	1/14/2006	EMERGENCY	ABNORMAL BODY MOVEMENTS	BROTHER IN LAW	2 D	HOME	BROTHER IN LAW	AWAKE		PLAYING	PLAYING
6	R	15	M	8/7/2005	EMERGENCY	ABNORMAL BODY MOVEMENTS	GUARDIAN	1 D	HOME	GUARDIAN	AWAKE		PLAYING	PLAYING
7	V	14	M	11/6/2005	OPD	ABNORMAL BODY MOVEMENTS	FATHER	FEW HOURS	HOME	PARENTS	SLEEPING	>10 MIN		
8	HS	12	M	10/16/2008	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	4 D	HOME	PARENTS	SLEEPING	>10 MIN	WATCHING MOVIE	WATCHING MOVIE
9	P	11	F	12/28/2009	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	9 D	HOME	FATHER	AWAKE		WATCHING MOVIE	WATCHING MOVIE
10	M	11	M	1/25/2009	EMERGENCY	ABNORMAL BODY MOVEMENTS	MOTHER	1 D	HOSPITAL	MOTHER	AWAKE		WATCHING MOVIE	WATCHING MOVIE
11	SS	11	M	11/28/2008	EMERGENCY	ABNORMAL BODY MOVEMENTS	MOTHER	2 D	HOME	MOTHER	AWAKE		PLAYING	PLAYING
12	KV	13	M	9/20/2006	OPD	ABNORMAL BODY MOVEMENTS	FATHER	FEW MONTHS	HOME	FATHER	SLEEPING	>10 MIN		
13	PG	16	M	2/18/2004	OPD	ABNORMAL BODY MOVEMENTS	FATHER	6 D	HOME	PARENTS	AWAKE		TAKING BATH	TAKING BATH
14	BS	15	M	9/27/2004	EMERGENCY	ABNORMAL BODY MOVEMENTS	FATHER	2 D	HOME	PARENTS	SLEEPING			
15	BS	16	M	6/3/2004	EMERGENCY	FEVER,COUGH,THROAT PAIN	UNCLE	1 D	HOSPITAL	STAFF /RELATIVES	SLEEPING			
16	G	12	M	8/12/2008	EMERGENCY	IN INTUBATED STATE	FATHER	2 D	HOSPITAL	FATHER	SLEEPING			
17	A	12	F	8/30/2008	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	1 D	HOME	MOTHER	AWAKE		TAKING BATH	TAKING BATH
18	S	11	M	7/30/2008	EMERGENCY	ABNORMAL BODY MOVEMENTS	HIMSELF	1 D	HOME	YOUNGER BROTHER	AWAKE		EATING FOOD	EATING FOOD
19	G	17	M	3/17/2003	EMERGENCY	ABNORMAL BODY MOVEMENTS	FATHER	1D	HOME	PARENTS	AWAKE		WATCHING MOVIE	WATCHING MOVIE
20	G	11	F	5/20/2010	OPD	ABNORMAL BODY MOVEMENTS	FATHER/MOTHER	6 D	HOME	MOTHER	SLEEPING	>`10 MIN		
21	MA	13	M	2/6/2007	EMERGENCY	ABNORMAL BODY MOVEMENTS	FATHER	1 D	HOME	FATHER	AWAKE		EATING FOOD	EATING FOOD
22	AK	11	F	2/8/2009	EMERGENCY	TEMPORARY DARKNESS OVER EYES	FATHER	2 D	HOME	PARENTS	AWAKE		WATCHING MOVIE	WATCHING MOVIE
23	ES	10	M	2/8/2010	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	1 MONTH BACK	HOME	MOTHER	AWAKE		JUST AWAKE	
24	SS	15	M	9/24/2005	OPD	ABNORMAL BODY MOVEMENTS	HIMSELF	1 D	HOME	PARENTS	AWAKE		WATCHING MOVIE	WATCHING MOVIE
25	HR	15	M	6/8/2005	EMERGENCY	P/O/C/O DECOMPRESSIVE FRONTOTEMPORAL CRANIECTOMY WITH SDH	MOTHER	FEW MINUTES	HOSPITAL	DOCTORS	SLEEPING		UNCONSCIOUS	
26	MD	10	M	10/10/2010	EMERGENCY	HEADACHE,ALTERED SENSORIUM ,LOC	PARENTS	3 HOURS	HOME	PARENTS	AWAKE		PLAYING	PLAYING
27	JS	10	M	8/12/2010	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	1 D	HOME	PARENTS	SLEEPING	<5 MINUTES		
28	SU	13	M	5/15/2007	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	1 MONTH BACK	HOME	PARENTS	SLEEPING	<5 MINUTES		
29	GS	10	M	10/8/2009	OPD	ABNORMAL BODY MOVEMENTS	FATHER	1 MONTH BACK	HOME	PARENTS	SLEEPING		GOING TO SLEEP	GOING TO SLEEP
30	SD	13	M	11/27/2007	EMERGENCY	ABNORMAL BODY MOVEMENTS	MOTHER	1 D	HOME	PARENTS	SLEEPING	<5 MINUTES		
31	P	12	M	5/10/2008	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	1 D	HOME	MOTHER	SLEEPING	>10 MIN		
32	G	13	M	3/19/2009	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	4 D	HOME	MOTHER	AWAKE		TAKING BATH	TAKING BATH
33	P	13	F	9/25/2007	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	1 D	HOME	MOTHER	AWAKE		PLAYING	PLAYING
34	JS	14	F	6/14/2007	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	15 D	HOME	MOTHER	AWAKE		EATING FOOD	EATING FOOD
35	S	11	M	8/15/2009	OPD	ABNORMAL BODY MOVEMENTS	MASI	1 WK	SCHOOL	TEACHER	AWAKE		STUDYING	STUDYING
36	NA	12	M	10/6/2009	OPD	ABNORMAL BODY MOVEMENTS	FATHER	3 D	HOME	FATHER	AWAKE		GOING TO SLEEP	GOING TO SLEEP
37	G	13	M	2/23/2009	EMERGENCY	ABNORMAL BODY MOVEMENTS	MOTHER	1 D	HOSPITAL	MOTHER	SLEEPING	>1 HOUR		

Sr. No	NAME	Pre-ictus (Aura)	Describe	Duration	Generalized onset Motor	Generalized onset Non-motor	FOCAL ONSET	Focal onset Motor	Focal onset Non-motor	Unknown onset Motor	Unknown onset Non-motor	Unclassified	Uprolling of eye balls	Bowel/ Bladder Incontinence
1	KS	NONE								MOTOR/CLONIC				
2	GS	NONE			TONIC-CLONIC									
3	A	NONE			TONIC									
4	DK	VISUAL	DARKNESS ON ONE SIDE	< 5 MINUTES			IMPAIRED AWARENESS	CLONIC					PRESENT	
5	GS	NONE			TONIC-CLONIC								PRESENT	PRESENT
6	R	NONE			TONIC-CLONIC									
7	V	NONE								MOTOR/CLONIC			PRESENT	PRESENT
8	HS	NONE			TONIC -CLONIC								PRESENT	PRESENT
9	P	NONE					IMPAIRED AWARENESS	CLONIC					PRESENT	
10	M	NONE			TONIC -CLONIC								PRESENT	
11	SS	VERTIGINOUS	VERTIGO				AWARE	CLONIC						
12	KV	NONE			TONIC CLONIC								PRESENT	
13	PG	VERTIGINOUS	VERTIGO	<5 MINUTES	MYOCLONIC -TONIC -CLONIC									
14	BS	VERTIGINOUS	VERTIGO		TONIC CLONIC									
15	BS	NONE			TONIC CLONIC			`						
16	G	NONE			TONIC									
17	A	NONE					IMPAIRED AWARENESS	CLONIC			BEHAVIOURAL ARREST PRESENT		PRESENT	
18	S	VERTIGINOUS	VERTIGO	> 10 MINUTES			IMPAIRED AWARENESS	CLONIC						
19	G	NONE			TONIC CLONIC								PRESENT	PRESENT
20	G	NONE			TONIC CLONIC									
21	MA	NONE			TONIC CLONIC									
22	AK	NONE					IMPAIRED AWARENESS		SENSORY(VISUAL)					
23	ES	GUSTATORY	SALIVATION	5-10 MINUTES			AWARE	CLONIC						
24	SS	AUDITORY	AUDITORY	<5 MINUTES			IMPAIRED AWARENESS	TONIC			BEHAVIOURAL ARREST PRESENT		PRESENT	PRESENT
25	HR	NONE					IMPAIRED AWARENESS	TONIC						
26	MD	VERTIGINOUS	HEADACHE	> 1 HOUR							BEHAVIOURAL ARREST PRESENT			PRESENT
27	JS	VERTIGINOUS	VERTIGO	< 5 MINUTES	TONIC CLONIC									
28	SU	VERTIGINOUS	HEADACHE	5-10 MINUTES			AWARE	TONIC					PRESENT	
29	GS	NONE			TONIC CLONIC								PRESENT	PRESENT
30	SD	AUTONOMIC	WEAKNESS OF LIMBS		TONIC									
31	P	NONE			TONIC CLONIC								PRESENT	PRESENT
32	G	NONE					AWARE		SENSORY (VISUAL)				PRESENT	
33	P	NONE			TONIC								PRESENT	
34	JS	NONE			TONIC CLONIC								PRESENT	PRESENT
35	S	VERTIGINOUS	VERTIGO	5-10 MINUTES	TONIC CLONIC								PRESENT	
36	NA	VERTIGINOUS	NAUSEA	< 5 MINUTES	CLONIC								PRESENT	
37	G	VISUAL	DARKNESS ON ONE SIDE	<5 MINUTES			IMPAIRED AWARENESS		SENSORY (VISUAL)					

Sr. No	NAME	Tongue bite	Duration	Post-ictal	Post-ictal Duration	Total duration of seizure	Clustering of seizures CLUSTERING:Present/ absent	Clustering of seizures Describe	Does the patient have more than one kind of seizures?	Has someone video-recorded the seizure episodes?	What was done at the time of seizure?	Review of systems	Family History OF SEIZURE	Past Medical History
1	KS		<5 MIN	HEADACHE/SLEEPINESS	<5 MIN	<5 MIN	ABSENT		N	N	TRIED TO GIVE WATER	WNL	NONE	NONE
2	GS		<5 MIN	NONE		5-30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	K/C/O CELIAC DISEASE
3	A	PRESENT	5-10 MIN	CONFUSION/AMNESIA/HEADACHE	5-10 MIN	5-30 MIN	ABSENT		N	N	TRIED TO GIVE WATER	WNL	NONE	None
4	DK		5-10 MIN	CONFUSION/AMNESIA/SLEEPINESS/VOMITING	5-10 MIN	5-30 MIN	ABSENT		N	N	TRIED TO GIVE WATER	WNL	NONE	NONE
5	GS	PRESENT	<5 MIN	CONFUSION/ABNORMAL BEHAVIOUR	<5 MIN	5-30 MIN	PRESENT	4 EPISODES	N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	H/o osteomyelitis 7 years
6	R		>1 HOUR	NONE		MORE THAN 30 MIN	PRESENT	CONTINUOUS	N	Y	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
7	V	PRESENT	5-10 MIN	CONFUSION	5-10 MIN	5-30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	PRESENT	NONE
8	HS		<5 MIN	CONFUSION/SLEEPINESS	5-10 MIN	5-30 MIN	PRESENT	2 EPISODES	N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	OPERATED
9	P		>10 MIN	CONFUSION/AMNESIA	>10 MIN	5-30 MIN	PRESENT	2 EPISODES	Y	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
10	M		<5 MIN	CONFUSION/AMNESIA		<5 MIN	ABSENT		N	N	PUT FINGER IN MOUTH	ABNORMAL	NONE	NONE
11	SS		5-10 MIN	NONE		5-30 MIN	ABSENT		N	N	NONE	WNL	PRESENT	NONE
12	KV		5-10 MIN	SLEEPINESS	>10 MIN	5-30 MIN	ABSENT		N	N	PUT FINGER IN MOUTH	WNL	NONE	H/O INFECTIONS
13	PG		<5 MIN	NONE		<5 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
14	BS		5-10 MIN	CONFUSION , SLEEPINESS	>10 MIN	5-30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
15	BS		<5 MIN	NONE		<5 MIN	ABSENT		N	N	NONE	WNL	NONE	NONE
16	G		<5 MIN	NONE		<5 MIN	ABSENT		N	N	LOADED WITH MIDAZOLAM	WNL	NONE	NONE
17	A		5-10 MIN	HEADACHE		5-30 MIN	PRESENT	2 EPISODES	N	N	TRIED TO GIVE WATER	WNL	NONE	NONE
18	S		5-10 MIN	CONFUSION, SLEEPINESS	>10 MIN	5-30 MIN	ABSENT		N	N	MASSAGING FEET AND HANDS	WNL	NONE	H/O INFECTIONS
19	G		>10 MIN	HEADACHE,VOMITING	< 5 MIN	5-30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
20	G		>10 MIN	HEADACHE	>1 HOUR	MORE THAN 30 MIN	PRESENT	2 EPISODES	N	N	MASSAGING FEET AND HANDS	WNL	NONE	NONE
21	MA		<5 MIN	HEADACHE	>10 MIN	5-30 MIN	ABSENT		N	N	TRIED TO GIVE WATER	WNL	NONE	NONE
22	AK		<5 MIN			<5 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
23	ES		>10 MIN	NONE		MORE THAN 30 MIN	PRESENT	CONTINUOUS	N	N	TRIED TO GIVE WATER	WNL	NONE	NONE
24	SS		5-10 MIN	HEADACHE	<5 MIN	5-30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
25	HR		<5 MIN	CONFUSION	<5 MIN	5-30 MIN	ABSENT		N	N	LOADED WITH MIDAZOLAM	ABNORMAL	NONE	NONE
26	MD		>10 MIN	VOMITING	>10 MIN	MORE THAN 30 MIN	ABSENT		N	N	TRIED TO GIVE WATER	ABNORMAL	NONE	SIMILAR EPISODE AT 5 MONTHS
27	JS		<5 MIN	SLEEPINESS	5-10 MIN	5-30 MIN	ABSENT		N	N	TRIED TO GIVE WATER	WNL	NONE	H/O INFECTIONS
28	SU		>10 MIN	HEADACHE	>10 MIN	MORE THAN 30 MIN	ABSENT		N	N	TRIED TO GIVE WATER	WNL	NONE	NONE
29	GS	PRESENT	>10 MIN	CONFUSION	>10 MIN	MORE THAN 30 MIN	ABSENT		N	N	TRIED TO GIVE WATER	WNL	NONE	NONE
30	SD		<5 MIN	NONE		<5 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
31	P	PRESENT	>10 MIN	UNCONSCIOUS	>10 MIN	MORE THAN 30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	PRESENT	NONE
32	G		>10 MIN	NONE		MORE THAN 30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	PRESENT	H/O FEBRILE SEIZURES
33	P	PRESENT	<5 MIN	NONE		<5 MIN	ABSENT		N	N	KEPT AT HOME	WNL	PRESENT	NONE
34	JS	PRESENT	5-10 MIN	NONE		MORE THAN 30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
35	S		<5 MIN	CONFUSION	>1 HOUR	MORE THAN 30 MIN	PRESENT	3 EPISODES	N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
36	NA		5-10 MIN	NONE		5-30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
37	G		<5 MIN	SLEEPINESS	>1 HOUR	MORE THAN 30 MIN	ABSENT		N	N	LOADED WITH MIDAZOLAM	WNL	NONE	NONE

Sr. No	NAME	Medications	Birth history	Development	Wt	Ht	General Physical Exam	Dysmorphism	Neurocutaneous signs	Systemic Exam	Neurological Exam	AWAKE EEG	SLEEP EEG	CT	MRI	Calcium	Final Diagnosis Acute symptomatic seizure	Final Diagnosis Unprovoked seizure
1	KS	NONE	NONE	NORMAL	29.7	137.5	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ND	ABNORMAL	NORMAL		GENETIC
2	GS	NONE	NONE	NORMAL	35	147	WNL	ABSENT	NONE	WNL	ABNORMAL	ND	ND	ND	ABNORMAL	NORMAL	ENCEPHALITIS	
3	A	NONE	NONE	NORMAL	50	154	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	WNL	WNL	NORMAL		UNKNOWN
4	DK	NONE	NNH	NORMAL	38	144	WNL	ABSENT	NONE	WNL	WNL	ABNORMAL	ABNORMAL	WNL	ABNORMAL	NORMAL		STRUCTURAL
5	GS	NONE	NONE	NORMAL	45	108	WNL	ABSENT	NONE	WNL	WNL	ND	ND	ABNORMAL	ABNORMAL	NORMAL	HEAD INJURY	
6	R	NONE	NONE	NORMAL	50	134	WNL	ABSENT	NONE	WNL	WNL	ND	ND	WNL	WNL	NORMAL		UNKNOWN
7	V	NONE	NONE	NORMAL	53	164	WNL	ABSENT	NONE	WNL	WNL	ABNORMAL	ABNORMAL	ND	ABNORMAL	NORMAL		GENETIC
8	HS	NONE	NONE	ABNORMAL	30	148	WNL	PRESENT	NONE	WNL	WNL	ND	ND	ND	ABNORMAL	LESS	HYPOCALCEMIA	
9	P	NONE	NONE	NORMAL	41.5	146	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ND	WNL	NORMAL		GENETIC
10	M	NONE	NNH	ABNORMAL	35	149	WNL	ABSENT	NONE	WNL	ABNORMAL	ND	ND	ND	WNL	LESS	HYPOCALCEMIA	
11	SS	NONE	NONE	NORMAL	48	154	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ABNORMAL	ABNORNAL	NORMAL	INFECTIOUS(NCC)	
12	KV	NONE	NONE	NORMAL	54	154	WNL	ABSENT	NONE	WNL	WNL	ND	WNL	ABNORMAL	ABNORMAL	NORMAL	INFECTIOUS(NCC)	
13	PG	NONE	NONE	NORMAL	54	154	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	ND	WNL	NORMAL		GENETIC
14	BS	NONE	NONE	NORMAL	58	156	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	ND	ABNORMAL	NORMAL	INFECTIOUS (NCC)	
15	BS	NONE	NONE	NORMAL	53	160	WNL	ABSENT	NONE	ABNORMAL	WNL	ND	ND	WNL	ND	NORMAL	ENCEPHALITIS	
16	G	NONE	NONE	NORMAL	36	144	WNL	ABSENT	NONE	WNL	ABNORMAL	WNL	WNL	ABNORMAL	ND	NORMAL	HEAD INJURY	
17	A	NONE	NONE	NORMAL	40	146	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ND	WNL	NORMAL		UNKNOWN
18	S	NONE	NONE	NORMAL	31.8	142	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	ND	WNL	NORMAL		GENETIC
19	G	NONE	NONE	NORMAL	44	148	WNL	ABSENT	NONE	WNL	ABNORMAL	ND	ND	ND	ABNORMAL	NORMAL	TUBERCULAR MENINGITIS	
20	G	NONE	NONE	NORMAL	38	148	WNL	ABSENT	NONE	WNL	WNL	ND	ND	ND	ABNORMAL	NORMAL	MENINGITIS	
21	MA	NONE	NONE	NORMAL	64	160	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ND	WNL	NORMAL		GENETIC
22	AK	NONE	NONE	NORMAL	44	120	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	ND	ABNORMAL	NORMAL		STRUCTURAL
23	ES	ONGOING PHNYTOIN	NONE	NORMAL	24.7	131	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ABNORMAL	ND	NORMAL	INFECTIOUS (NCC)	
24	SS	ONGOING VALPROATE	NONE	NORMAL	51.7	167	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	ND	WNL	NORMAL		GENETIC
25	HR	NONE	NONE	NORMAL	60	160	HEMIPARALYSIS	ABSENT	NONE	WNL	ABNORMAL	ND	ND	ABNORMAL	ND	NORMAL	HEAD INJURY	
26	MD	NONE	NONE	NORMAL	30	128	WNL	ABSENT	NONE	WNL	ABNORMAL	ND	ND	ABNORMAL	ABNORMAL	NORMAL	HEAD INJURY	
27	JS	NONE	NONE	NORMAL	39	135	WNL	ABSENT	NONE	WNL	WNL	ABNORMAL	ABNORMAL	ND	WNL	NORMAL		GENETIC
28	SU	NONE	NONE	NORMAL	42	120	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ND	WNL	NORMAL		GENETIC
29	GS	NONE	NONE	NORMAL	42	120	WNL	ABSENT	NONE	WNL	WNL	ND	ABNORMAL	ND	WNL	NORMAL		GENETIC
30	SD	NONE	NONE	NORMAL	54	170	WNL	ABSENT	NONE	WNL	WNL	ND	ND	ND	ND	LESS	HYPOCALCEMIA	
31	P	NONE	NONE	NORMAL	51	158	WNL	ABSENT	NONE	WNL	WNL	ABNORMAL	ND	WNL	WNL	NORMAL		GENETIC
32	G	NONE	PREMATURITY	NORMAL	37	147	WNL	ABSENT	NONE	WNL	WNL	ABNORMAL	ABNORMAL	WNL	ND	NORMAL		GENETIC
33	P	ACETAZOLAMIDE	NONE	NORMAL	45	160	WNL	ABSENT	NONE	WNL	WNL	ABNORMAL	ND	ND	WNL	NORMAL		GENETIC
34	JS	FLUOXETINE	NONE	NORMAL	38	135	WNL	ABSENT	NONE	WNL	WNL	ABNORMAL	ND	ND	ND	NORMAL		UNKNOWN
35	S	NONE	NONE	NORMAL	35.5	154	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	ND	ABNORMAL	NORMAL		GENETIC
36	NA	NONE	PREMATURITY	NORMAL	42	152	WNL	ABSENT	NONE	WNL	WNL	ABNORMAL	ND	ND	WNL	NORMAL		GENETIC
37	G	NONE	NONE	NORMAL	45	154	WNL	ABSENT	NONE	WNL	WNL	ND	ND	ND	ND	NORMAL		UNKNOWN

Sr. No	NAME	AGE	GENDER	DOB	AREA OF PRESENTATION	CHIEF COMPLAINT	NARRATOR	DURATION SINCE SEIZURE	PLACE OF OCCURRENCE	SEIZURE WITNESSED BY	CIRCUMSTANCE OF OCCURRENCE	Time of the day IF IN SLEEP,TIME SINCE SLEEP	Time of the day If awake, what was child doing	Pro-ictus (Triggers)
38	DS	16	M	2/4/2020	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	6 D	HOME	MOTHER	SLEEPING	>1 HOUR		
39	KS	16	M	11/12/2004	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	1 D	HOME	PARENTS	SLEEPING	>1 HOUR		
40	AM	13	M	12/20/2007	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	3 D	HOME	PARENTS	AWAKE		PLAYING	PLAYING
41	D	15	F	6/27/2006	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	2 D	HOME	MOTHER	AWAKE		JUST AWAKE	
42	B	10	M	6/10/2009	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	2D	HOME	PARENTS	AWAKE		STUDYING	STUDYING
43	A	13	F	10/4/2007	OPD	ABNORMAL BODY MOVEMENTS	MOTHER	2 D	HOME	MOTHER	AWAKE		JUST AWAKE	
44	GS	15	M	1/12/2010	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	1 D	HOME	PARENTS	AWAKE		WATCHING MOVIE	WATCHING MOVIE
45	BK	15	M	10/16/2004	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	1 D	SCHOOL	PARENTS	AWAKE		STUDYING	STUDYING
46	S	13	F	5/25/2007	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	1 D	HOME	PARENTS	SLEEPING	> 10 MINUTES		
47	R	10	F	12/3/2009	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	1 D	HOME	PARENTS	AWAKE		WATCHING MOVIE	WATCHING MOVIE
48	R	17	M	9/24/2003	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	1 D	HOME	PARENTS	SLEEPING	> 1 HOUR		
49	AS	14	M	4/12/2007	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	2 D	HOME	PARENTS	SLEEPING	>1 HOUR		
50	K	16	M	11/12/2004	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	1 D	HOME	PARENTS	SLEEPING	>1 HOUR		
51	PK	12	M	5/7/2009	OPD	ABNORMAL BODY MOVEMENTS	PARENTS	1 D	HOME	PARENTS	AWAKE		JUST AWAKE	

Sr. No	NAME	Pre-ictus (Aura)	Describe	Duration	Generalized onset Motor	Generalized onset Non-motor	FOCAL ONSET	Focal onset Motor	Focal onset Non-motor	Unknown onset Motor	Unknown onset Non-motor	Unclassified	Uprolling of eye balls	Bowel/ Bladder Incontinence
38	DS	NONE			TONIC-CLONIC									
39	KS	NONE			TONIC CLONIC									
40	AM	NONE			MYOCLONUS									
41	D	NONE			MYOCLONUS								PRESENT	
42	B	NONE			TONIC CLONIC									
43	A	VERTIGINOUS	NAUSEA											PRESENT
44	GS	VERTIGINOUS	DARKNESS ON ONE SIDE	<5 MINUTES			IMPAIRED AWARENESS	TONIC						
45	BK	NONE			TONIC CLONIC								PRESENT	PRESENT
46	S	NONE					IMPAIRED AWRENESS	TONIC						
47	R	NONE			TONIC									
48	R	NONE			TONIC CLONIC								PRESENT	PRESENT
49	AS	NONE			TONIC CLONIC								PRESENT	PRSEENT
50	K	NONE			TONIC CLONIC								PRESENT	PRESENT
51	PK	NONE			MYOCLONUS									PRESENT

Sr. No	NAME	Tongue bite	Duration	Post-ictal	Post-ictal Duration	Total duration of seizure	Clustering of seizures CLUSTERING:Present/ absent	Clustering of seizures Describe	Does the patient have more than one kind of seizures?	Has someone video-recorded the seizure episodes?	What was done at the time of seizure?	Review of systems	Family History OF SEIZURE	Past Medical History
38	DS		>10 MIN	SLEEPINESS	>1 HOUR	MORE THAN 30 MIN	PRESENT	2 EPISODES	N	N	MASSAGING FEET AND HANDS	WNL	NONE	NONE
39	KS	PRESENT	5-10 MIN	NONE		5-30 MIN	ABSENT		N	N	MASSAGING FEET AND HANDS	WNL	NONE	NONE
40	AM		<5 MIN	NONE		5- 30 MIN	PRESENT	2 EPISODES	N	Y	KEPT AT HOME	WNL	NONE	NONE
41	D		<5 MIN	CONFUSION/HEADACHE	>1 HOUR	MORE THAN 30 MIN	ABSENT		N	N	TRIED TO GIVE WATER	WNL	NONE	NONE
42	B		>10 MIN	NONE		MORE THAN 30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
43	A		>10 MIN	NONE		MORE THAN 30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
44	GS		<5 MIN	HEADACHE/SLEEPINESS	>1 HOUR	MORE THAN 30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
45	BK	PRESENT	>10 MIN	CONFUSION, SLEEPINESS	>10 MIN	MORE THAN 30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	H/O SIMILAR EPISODE AT 1 YEAR
46	S		5-10 MIN	CONFUSION , SLEEPINESS	>1 HOUR	MORE THAN 30 MIN	PRESENT	3 EPISODES	N	N	TRIED TO GIVE WATER	WNL	NONE	NONE
47	R		5-10 MIN	CONFUSION/HEADACHE	>10 MIN	MORE THAN 30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	OPERATED
48	R	PRESENT	5-10 MIN	NONE		5-30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	H/O MIGRAINE
49	AS	PRESENT	<5 MIN	AMNESIA/SLEEPINESS	>10 MIN	MORE THAN 30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE
50	K	PRESENT	<5 MIN	NONE		<5 MIN	ABSENT		N	N	MASSAGING FEET AND HANDS	WNL	NONE	NONE
51	PK		<5 MIN	CONFUSION/SLEEPINESS	5-10 MIN	5-30 MIN	ABSENT		N	N	BROUGHT TO THE HOSPITAL	WNL	NONE	NONE

Sr. No	NAME	Medications	Birth history	Development	Wt	Ht	General Physical Exam	Dysmorphism	Neurocutaneous signs	Systemic Exam	Neurological Exam	AWAKE EEG	SLEEP EEG	CT	MRI	Calcium	Final Diagnosis Acute symptomatic seizure	Final Diagnosis Unprovoked seizure
38	DS	NONE	NONE	NORMAL	38	140	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	ND	WNL	NORMAL		UNKNOWN
39	KS	NONE	PREMATURITY	NORMAL	54	154	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	WNL	WNL	NORMAL		GENETIC
40	AM	NONE	NONE	NORMAL	31.5	140	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ND	ND	NORMAL		UNKNOWN
41	D	NONE	NONE	NORMAL	42	146	WNL	ABSENT	NONE	WNL	WNL	WNL	ABNORMAL	WNL	ND	NORMAL		GENETIC
42	B	NONE	NONE	NORMAL	45	140	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ND	WNL	NORMAL		GENETIC
43	A	NONE	NONE	NORMAL	41	139	WNL	ABSENT	NONE	WNL	WNL	WNL	ABNORMAL	ND	WNL	NORMAL		UNKNOWN
44	GS	NONE	NNH	NORMAL	40	144	WNL	ABSENT	NONE	WNL	WNL	WNL	ABNORMAL	ND	WNL	NORMAL		STRUCTURAL
45	BK	NONE	NONE	NORMAL	64	154	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	ND	WNL	LESS	HYPOCALCEMIA	
46	S	NONE	NONE	NORMAL	39	140	WNL	ABSENT	NONE	WNL	WNL	ND	ABNORMAL	ND	WNL	NORMAL		GENETIC
47	R	NONE	NONE	NORMAL	29	144	WNL	ABSENT	NONE	WNL	WNL	ND	ABNORMAL	ND	WNL	NORMAL		GENETIC
48	R	NONE	NONE	NORMAL	68	154	WNL	ABSENT	NONE	WNL	WNL	ND	WNL	ND	WNL	NORMAL		GENETIC
49	AS	NONE	NONE	NORMAL	56	150	WNL	ABSENT	NONE	WNL	WNL	ND	WNL	ND	ABNORMAL	NORMAL		STRUCTURAL
50	K	NONE	PREMATURITY	NORMAL	58	151	WNL	ABSENT	NONE	WNL	WNL	WNL	WNL	ND	WNL	NORMAL		GENETIC
51	PK	NONE	NONE	NORMAL	35	140	WNL	ABSENT	NONE	WNL	WNL	WNL	ND	ND	WNL	NORMAL		GENETIC

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Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of **Dr. Tanvi**, a student of your college for **MD Paediatrics** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
New onset seizures in adolescents : a prospective observational study

Supervisor
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The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

S P Singh
Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. ***If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.***
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

NEW ONSET SEIZURES IN ADOLESCENTS: A PROSPECTIVE OBSERVATIONAL STUDY

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN

PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

In

PAEDIATRICS

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

NOVEMBER, 2019

Dr. Tanvi

DEPARTMENT OF PAEDIATRICS
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

TITLE	NEW ONSET SEIZURES IN ADOLESCENTS: A PROSPECTIVE OBSERVATIONAL STUDY
DEGREE	MD Paediatrics
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CO – SUPERVISOR	Dr. Deepak Bhat, Professor, Department of Pediatrics, DMC&H, Ludhiana
INSTITUTION	DAYANAND MEDICAL COLLEGE AND HOSPITAL, LUDHIANA
UNIVERSITY	BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

Introduction: A seizure is a sudden disruption of the brain's normal electrical activity accompanied by altered consciousness and/or other neurological and behavioral manifestations. The International League Against Epilepsy (ILAE) classifies seizures into two major groups: focal or generalized, according to presence or absence of localized or focal onset, with each group having further subgroups. From etiological point of view, seizures are classified into provoked or unprovoked. Seizures caused by acute brain insults secondary to fever, head injury, neuro-infections, exposure to toxins or drugs, acute metabolic derangements such as hypoglycemia, hypocalcemia, hypo-/hypernatremia are called provoked or acute symptomatic seizures. Seizures can also be the first manifestation of epilepsy which may be genetic in origin or caused by structural abnormalities in the brain related to previous brain insults or other etiologies like structural malformations, brain tumors etc. These are also called unprovoked seizures and represent true epileptic seizures.

AIM OF STUDY: 1. To study clinical characteristics of first onset seizures, both provoked and unprovoked, in adolescents. 2) To determine the etiologies of first onset seizures in adolescents. 3) To study the electroencephalography (EEG) and MR neuroimaging characteristics of first onset seizures in adolescents

Materials & methods: This will be a prospective observational study on adolescent children presenting with new onset seizures to the pediatric department of DMCH, Ludhiana

Clinical Significance: Approximately 8-10 % people will experience a seizure in their lifetime. Only about 2% to 3% of patients go on to develop epilepsy. Understanding the underlying etiology leading to an accurate diagnosis is necessary to ensure appropriate treatment and that patients with low risk for recurrence are not treated unnecessarily.

Keywords: new-onset seizure, acute symptomatic seizures, epilepsy, adolescents

Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"NEW ONSET SEIZURES IN ADOLESCENTS: A PROSPECTIVE OBSERVATIONAL STUDY"** exist in the Department of Paediatrics, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate herself under our supervision and guidance.

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APPROVAL PROFORMA
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3	COURSE OF STUDY AND SUBJECT	MD. Paediatrics
4	DATE OF ADMISSION OF COURSE	6th May 2019
5	TITLE OF THE TOPIC	New Onset Seizures in Adolescents: A Prospective Observational Study

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

A seizure is a sudden disruption of the brain's normal electrical activity accompanied by altered consciousness and/or other neurological and behavioral manifestations.[1] The International League Against Epilepsy (ILAE) classifies seizures into two major groups: focal or generalized, according to presence or absence of localized or focal onset, with each group having further subgroups.[2] From etiological point of view, seizures are classified into provoked or unprovoked. Seizures caused by acute brain insults secondary to fever, head injury, neuro-infections, exposure to toxins or drugs, acute metabolic derangements such as hypoglycemia, hypocalcemia, hypo-/hypernatremia are called provoked or acute symptomatic seizures.[3] Seizures can also be the first manifestation of epilepsy which may be genetic in origin or caused by structural abnormalities in the brain related to previous brain insults or other etiologies like structural malformations, brain tumors etc.[4] These are also called unprovoked seizures and represent true epileptic seizures.

Seizures are a common manifestation of neurological disorders in adolescents and like at any other age, be related to acute brain insult or cause by epileptic predisposition.[5] Common causes of acute symptomatic seizures in adolescents include traumatic brain injury, neuro-infections such as meningitis, encephalitis, exposure to drugs including drugs of abuse, and metabolic derangements.[2,3] Epilepsy during adolescents in majority of the cases is genetic in origin but may be fist manifestation of old brain injury sustained during childhood. Brain tumors can also manifest with seizures.[5] Neurocysticercosis has emerged as a common cause of new onset seizures in children and adults in India.[6,7] The frequency and spectrum of disorders associated with occurrence of seizures in adolescents is different than that seen in children and adults. It will be of great interest to study the epileptology of first onset seizures in adolescents

The Advantages of enteral nutrition are known, but in critically ill children admitted in pediatric ICU, there may be many factors leading to delay in initiation of enteral nutrition. These factors could be presence of shock, high inotropes, altered sensorium, on non invasive ventilation, vomiting, intolerance to feeds etc. Though there are many factors, but there is no prospective study available from region, where these factors are studied and also to determine affect of these factors on ultimate outcome of these critical ill patients.

Seizures are a common disorder in adolescents and can results from variety of causes. Acute brain insults secondary to head injury, neuroinfections, exposure to toxins or drugs, acute metabolic derangements such as hypoglycemia, hypocalcemia, hypo-/hypernatremia can cause seizures. These are called provoked or acute symptomatic seizures. Seizures in adolescents can also be the first manifestation of epilepsy (unprovoked seizures) which may be genetic in origin or caused by structural abnormalities in the brain related to previous brain insults or other etiologies like structural malformations, brain tumors etc. There is dearth of literature on the new onset seizures in adolescents in India, especially with regard to provoked and unprovoked seizures. The results of this study will provide the scientific community with the new knowledge in the epidemiological, epileptological, and etiological characteristics of the first onset seizures in adolescents in India. It is expected that the accrued knowledge ultimately will help the health planner and policy makers to devise appropriate preventive and therapeutic interventions.

6.2 REVIEW OF LITERATURE

New onset seizures can be acute symptomatic or provoked seizures, caused by acute brain insult secondary to trauma, neuroinfectious, drugs, toxic or metabolic etiologies.[3] These seizures account for nearly 40% of the total seizures.[8] The incidence of acute symptomatic seizures is 29–39 per 100,000 per year. The incidence of single unprovoked seizures is 23–61 per 100,000 person-years.[8] There is 3.6% overall life-time risk of developing an acute symptomatic seizure which is almost similar about 2-3% of epilepsy.[8] New onset unprovoked seizures constitute epileptic seizures and etiologically represent either genetic epilepsies or are caused by previous brain damage (remote symptomatic) caused by neuro-trauma, neuro-infections, cerebrovascular injuries, birth related brain injuries. Both type of seizures – acute symptomatic and unprovoked epileptic seizures occur in adolescents but there is not enough literature on the subject.

Narayanan and Murthy [9] studied acute symptomatic seizures in neurological intensive care unit over a period of 28 months. Acute symptomatic seizures accounted for 2.1% of all the admissions to neurological intensive care unit. Generalized as well as focal seizures were observed. Vast majority were due to central nervous system infections (32%), metabolic derangements (32%), and cerebrovascular disorders (21%). In another hospital-based study from South India, Murthy and Yangala [10] analyzed 572 patients with acute symptomatic seizures seen over 5-year period. Vast majority had focal seizures. Central nervous system infections were diagnosed in 145 (28%), accounted by neurotuberculosis (n=83), neurocysticercosis (n=50), pyogenic meningitis (n=5), brain abscess (n=3) and encephalitis (n=4). Cerebrovascular disorders accounted for 74 (14%) and tumors for 40 (7%) cases. It is to be noted that authors recognized single CT enhancing lesion (SCTEL) in 262 (50%) cases. Soni et al. [11] in 2017, from India prospectively evaluated 105 children (3-144 months) with acute symptomatic seizures and 82% of these cases were due to CNS infections. Meningitis and meningo-encephalitis accounted for majority

of the infections, followed by neur-tuberculosis. Number of adolescent children (>10 years of age) was likely small though not specified in the study. Similarly, a study by Idro and colleagues [12] from rural Kenya identified infections to be associated with acute symptomatic seizures in over 80% of the children, with malaria being the commonest. Number of children older than 10 years was not specified. In a study of acute symptomatic seizures in children from Taiwan, by Huang et al. [13] CNS infections (meningitis, encephalitis) accounted for majority (46.1%) of the cases, followed by intracranial hemorrhage (20.6%) and metabolic/toxic (11.1%) causes. There was significant variation according the age. Annegers et al. [14] found similar etiologies but with relative frequencies dependent upon age and gender to be responsible for acute symptomatic seizures. Overall, there was 3.6% risk of experiencing an acute symptomatic seizure. In this study, major causes of acute symptomatic seizures were traumatic brain injury, cerebrovascular diseases, drug withdrawal, and central nervous system infections. Lee and Kim [15] from South Korea described occurrence of acute symptomatic seizures in 109 children seen over 8 years. Acute gastroenteritis was identified as the cause in 33% followed by unspecified encephalopathy in 31.2%. Metabolic/toxic etiology was identified in 19.3%. Central nervous system infections accounted for only 11% of the cases. In a study from UK, Smith et al. [16] febrile seizures was the commonest diagnosis occurring in 75 (63%) children amongst 118 children who presented with first seizure. Thirteen children had acute symptomatic seizures secondary to various metabolic and neurological causes

Unprovoked seizures constitute the true epileptic seizures. The lifetime incidence of an afebrile epileptic seizure is 5%. The diagnosis of an epileptic seizure demands its differentiation from non-seizures events as well as exclusion of an acute symptomatic cause. The epileptic seizures etiologically are classified as remote symptomatic, denoting previous brain injury or idiopathic/genetic. Relative frequency of each category depends upon several factors including age, gender, geographical location, and laboratory studies. EEG abnormalities are

seen in about one-half patients after first seizure. Neuroimaging studies have yielded varied results depending whether or not children were included.[17] In this prospective study in children presenting with first unprovoked seizure, abnormalities were detected in 45 (21%) out of 218 imaging studies. Focal ischemic lesions and cerebral dysgenesis accounted for majority of the lesions.[17] Berg et al. [18] in 1999 reported 613 children up to 15 years of age with new onset epilepsy. Median age at the time of first seizure was 5.3 years. Boys and girls were equally distributed in the cohort. Only 84 (13.7%) children were aged more than 10 years. Focal seizures were more common than generalized seizures. Cryptogenic category was the most common etiology observed in 317 (51.7%) children, followed by idiopathic in 185 (30.2%). Remote symptomatic etiology was identified in 111 (18 %) children. An epilepsy syndrome was made in 609 children. Focal epilepsy syndrome was assigned to 58.6% children and 29.0% children had generalized epilepsy syndrome. Benign Rolandic epilepsy occurred in 10% of the cohort. Childhood absence epilepsy was the most common generalized epilepsy syndrome in this cohort. King et al. [19] studied 300 patients (aged 5-83 years) with new-onset unprovoked seizure with no apparent cause. There were 59 (20% of the total cases) children aged between 5 to 16 years. Focal or generalized epilepsy was diagnosed in 141 (47%) cases and 159 remained unclassified majority of whom has generalized seizures. After EEG and neuroimaging, a final diagnosis of epilepsy was achieved in 243 (81%). Most common specific generalized epilepsy syndrome were juvenile absence epilepsy and juvenile myoclonic epilepsy. Temporal lobe epilepsy was most common focal epilepsy syndrome seen in 57% of the cases of focal epilepsy. This was followed by benign Rolandic and benign occipital epilepsies seen in 7%.

A multicenter, prospective French study (CAROLE; Coordination Active du Reseau Observatoire Longitudinal de 1'Epilepsie) of patients (aged between 9-39 years; mean age 19 years; nearly 60% male)) with newly diagnosed unprovoked epileptic seizures found focal epilepsy in 46.2%, generalized seizures in 31.9%, and unclassified seizures in

21.9%. Generalized tonic clonic seizures were the most common type of generalized seizures. Etiologically, 24.6& had idiopathic etiology, and 57.8% had cryptogenic etiology. Symptomatic etiology was noted in 17.6%. Pre- and perinatal insult, cerebrovascular diseases, and head injury accounted for majority of the symptomatic cases.[20] Incidence of epilepsy and of all unprovoked seizures in the famous Rochester, study was 44 per 100,000 person-years, being higher in males than in females. The study included both children and adults, and the incidence was high in first years of life, and highest in persons 75 years of age or older. Cerebrovascular disease was the most common cause identified in 11% of cases. Mental retardation and/ or cerebral palsy was second common category of etiologies.[21] A recent prospective study of new onset seizures presenting as status epilepticus in children revealed 144 (10%) cases of status epilepticus out of 1,382 infants and children with new-onset seizures. Febrile seizures as the most common cause noted in 46 (32%). Most common acute symptomatic cause was CNS infection. Cerebral dysgenesis was the most common cause in remote symptomatic category. Number of adolescents in this study was not specified but was small.[22]

6.3 AIMS AND OBJECTIVES OF THE STUDY

1. To study the clinical and etiological characteristics of new onset seizures in adolescents.
2. To assess the spectrum of electroencephalographic and neuroimaging findings in adolescents with new onset seizures

7. MATERIALS AND METHODS

Study site: The study will be carried out in the Department of Pediatrics, Dayanand Medical College and Hospital, Ludhiana

Choice of Subjects: First seizure will be defined as a new onset seizure, focal or generalized, with no previous history of similar events. A cluster of seizures all of which occurring within 24 hours or the occurrence of status epilepticus at onset will be regarded as a single seizure. The seizure will be considered provoked when there is an immediate identifiable neurological insult such as fever, head trauma, drug/toxin exposure, a metabolic abnormality or meningitis/ encephalitis. The seizure will be considered unprovoked if there was no identifiable immediate neurological insult.

7.1 Source of Data

Adolescents attending the pediatric out-patient, in-patient and pediatric emergency will be the source of data. All adolescents with seizures or seizure-like presentation will undergo detailed historical interrogation to determine the nature of episodes. Those found to have non-epileptic spells will be excluded. Those who are found to have had a seizure will be enrolled if they met the inclusion criteria

7.2 METHOD OF COLLECTION OF DATA

Following inclusion and exclusion criteria will be employed:

Inclusion Criteria:

- New onset focal or generalized seizure. For unprovoked seizures, adolescents with prior history of neonatal, febrile, or other provoked seizures will not be excluded
- Adolescents presenting to the department within three months of onset of seizure will be enrolled
- Age 10 years to 18years
- Gender: either

Exclusion Criteria

- Age < 10 years, > 18 years
- Presentation three months after the onset of seizure
- Parents' refusal to participate.

The selected children meeting the inclusion criteria will undergo detailed clinical evaluation by history and physical examination. Laboratory investigations will be dictated by clinical suspicion of the underlying cause of seizures. Similarly, neuroimaging and electroencephalography (EEG) will be done on the basis of results of clinical evaluation.

Data will be prospectively collected on structure proforma

Duration of the study

This is a prospective study from Jan 2020 to March 2021

OUTCOME

We will find the common cause of acute symptomatic seizures and epileptic seizures in the adolescents.

Statistical Analysis

Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

NO investigation other than needed for routine care of the adolescents with new onset seizures as per standard protocol, will be done

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith

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PROFORMA

S.NO-

Name Age/Gender Date of Birth

MRD No. Place of presentation: OPD/IPD/ Emergency

Referring Diagnosis:

Chief complaint:

Narrator:

Duration:

Settings (Para-ictal):

Place of occurrence of seizure: Home/School/Others

Seizure witnessed by:

Circumstances of occurrence:

Awake/On Awakening/Drowsy/Sleeping

Time of the day

If in sleep, time since asleep:

If awake, what was child doing:

Signature of candidate :

Signature of consultant:

Pro-ictus (Triggers):

TV/videogames/working on computer/Mobile

Sleep deprivation/Alcohol/Drugs/Exams/

Pre-ictus (Aura):

None/Visual/auditory/sensory/olfactory/gustatory/vertiginous/psychic/autonomic

Describe:

Duration:

Per-Ictus:

Generalized onset

Motor: Tonic-clonic/Clonic/Tonic/Myoclonic/Myoclonic-tonic-clonic/

Myoclonic-atonic/Atonic/epileptic spasms

Non-motor:

Absences: Typical/atypical/myoclonic/eyelid myoclonia

Focal onset:

Aware/Impaired awareness

Motor: Automatism/Atonic/clonic/epileptic spasms/hyperkinetic/

myoclonic/tonic

Non-motor: Autonomic/behavior arrest/cognitive/emotional/sensory

Signature of candidate : **Signature of consultant:**

Focal to bilateral tonic clonic

Unknown onset:

Motor: tonic clonic/epileptic spasms

Non-motor: behavioral arrest

Unclassified

Duration:

Post-ictal: None/Confusion/amnesia/headache/sleepiness/vomiting/psychosis/

/abnormal behaviour/Todd's paralysis

Duration:

Total duration of seizure:

Clustering of seizures

Present/absent

Describe

Does the patient have more than one kind of seizures? No Yes (specify)

Has someone video-recorded the seizure episodes?

What was done at the time of seizure?

Signature of candidate :

Signature of consultant:

Any additional information if any

Review of systems:

Family History

Past Medical History

Medications

Birth history: Prematurity/HIE/neonatal jaundice/neonatal hypoglycemia/Meningitis/

encephalitis/Stroke/others

Development: Normal/Intellectual disability/Autism/others

Examination Wt Ht

General Physical Exam

Dysmorphism

Neurocutaneous signs

Systemic Exam

Neurological Exam

EEG

Signature of candidate :

Signature of consultant:

CT/MRI

Others:

Final Diagnosis:

Acute symptomatic seizure: Etiology

Unprovoked seizure: Genetic/Structural/Infectious/metabolic/Immune/Unknown

Signature of candidate :

Signature of consultant:

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....
am willing to participate in the study **"NEW ONSET SEIZURES IN ADOLESCENTS: A PROSPECTIVE OBSERVATIONAL STUDY"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

Name:

.....
Signature of the patient

Signature of Candidate

Signature of Supervisor

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"NEW ONSET SEIZURES IN ADOLESCENTS: A PROSPECTIVE OBSERVATIONAL STUDY"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

PATIENT'S PARENT/GUARDIAN CONSENT FORM

Date: ____/____/____

I .....F/o, M/o .....

R/o .....

am willing to let my child participate in the study entitled Bed **"NEW ONSET SEIZURES IN ADOLESCENTS: A PROSPECTIVE OBSERVATIONAL STUDY"**. The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason. This will not affect my treatment and investigation of my child.

Signature of the Witness	Signature/Thumb Impression of guardian
Name:	Name:

Signature of the Supervisor	Signature of Candidate

PATIENT'S PARENT/GUARDIAN CONSENT FORM

Date: ____/____/____

I .....F/o, M/o .....

R/o .....

am willing to let my child participate in the study entitled Bed **"NEW ONSET SEIZURES IN ADOLESCENTS: A PROSPECTIVE OBSERVATIONAL STUDY"**. The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason. This will not affect my treatment and investigation of my child.

_____	_____
Signature of the Witness	Signature/Thumb Impression of guardian
Name:	Name:

_____	_____
Signature of the Supervisor	Signature of Candidate

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"NEW ONSET SEIZURES IN ADOLESCENTS: A PROSPECTIVE OBSERVATIONAL STUDY" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____
ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____

ਪਤਾ _____

_____
ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____

ਪਤਾ _____

_____
ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____
ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ

मरीज के माता/पिता का अनुमति फार्म

तिथि ___/___/___

मैं _____ पिता/माता/_____
वासी _____,
अपने बच्चे के इस शोधकार्य में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ कि स्वीकृत देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है कि यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामूली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे बच्चे के इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर मरीज के माता/पिता के हस्ताक्षर

तिथि _____

पता _____

_____

_____

_____ _____

नरीक्षक के हस्ताक्षर प्रत्याक्षी के हस्ताक्षर

ਮਰੀਜ ਦੇ ਮਾਤਾ/ਪਿਤਾ ਦਾ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ __/___/____

ਮੈਂ _____ ਪਿਤਾ/ਮਾਤਾ _____,

ਵਾਸੀ _____

ਆਪਣੇ ਬੱਚੇ ਦੇ ਇਸ ਅਧਿਐਨ ਵਿੱਚ ਹਿੱਸਾ ਲੈਣ ਤੇ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿੱਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈਂ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈਂ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ/ਚਾਹੁੰਦੀ ਹਾਂ ਕਿ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂੰ ਕਿਸੇ ਵੀ ਤਰ੍ਹਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀਂ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂੰ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂੰ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂੰ ਇਸ ਖੋਜ ਵਿੱਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆਂ ਹੈ ਅਤੇ ਮੈਨੂੰ ਇਹ ਯਕੀਨ ਹੈ ਕਿ ਇਹ ਖਤਰਾ ਬਹੁਤਾ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂੰ ਇਸ ਖੋਜ ਕੰਮ ਵਿੱਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ-ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈਂ ਇਸ ਖੋਜ ਕੰਮ ਵਿੱਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇਂ ਵਾਪਸ ਲੈ ਸਕਦਾ/ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਬੱਚੇ ਦੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀਂ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ _____ ਮਿਤੀ _____

ਪਤਾ _____ ਪਤਾ _____

_____ _____

ਨਰੀਖਕ ਦੇ ਹਸਤਾਖਰ ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ